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Department of Labor

Occupational Safety and Health Administration

29 CFR Parts 1910, 1915, 1926, and 1928
Indoor Air Quality; Proposed Rule

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DEPARTMENT OF LABOR

Occupational Safety and Health Administration

29 CFR Parts 1910, 1915, 1926, 1928

[Docket No. H-122]

RIN 1218-AB37

Indoor Air Quality

AGENCY: Occupational Safety and Health Administration (OSHA), Labor.

ACTION: Notice of proposed rulemaking; notice of informal public hearing.

SUMMARY: By this notice, the Occupational Safety and Health Administration (OSHA) proposes to adopt standards addressing indoor air quality in indoor work environments. The basis for this proposed action is a preliminary determination that employees working in indoor work environments face a significant risk of material impairment to their health due to poor indoor air quality, and that compliance with the provisions proposed in this notice will substantially reduce that risk.

The provisions of the standard are proposed to apply to all indoor "nonindustrial work environments." In addition, all worksites, both industrial and nonindustrial within OSHA's jurisdiction are covered with respect to the proposed provisions addressing control of environmental tobacco smoke. The proposal would require affected employers to develop a written indoor air quality compliance plan and implement that plan through actions such as inspection and maintenance of building systems which influence indoor air quality.

Provisions under the standard also propose to require employers to implement controls for specific contaminants and their sources such as outdoor air contaminants, microbial contamination, maintenance and cleaning chemicals, pesticides, and other hazardous chemicals within indoor work environments. Designated smoking areas which are to be separate, enclosed rooms exhausted directly to the outside are proposed to be required in buildings where the smoking of tobacco products is not prohibited. Specific provisions are also proposed to limit the degradation of indoor air quality during the performance of renovation, remodeling and similar activities. Provisions for information and training of building system maintenance and operation workers and other employees within the facility are also included in this notice.

Finally, proposed provisions in this notice address the establishment, retention, availability, and transfer of records such as inspection and maintenance records, records of written compliance programs, and employee complaints of building-related illness.

The Agency invites the submission of written data, views and comments on all regulatory provisions proposed in this notice, and on all relevant issues pertinent to those provisions. OSHA is also scheduling an informal public hearing where persons may orally submit their views. It is noted here that subsequent Federal Register notices may be published subsequent to this notice, if the public presents views leading to a substantial change in focus or it is otherwise determined to be appropriate.

DATES: Comments on the proposed standard must be postmarked by June 29, 1994. Notices of intention to appear must be postmarked by June 20, 1994. Testimony and evidence to be submitted at the hearing must be postmarked by July 5, 1994. The hearing will commence at 9:30 a.m. on July 12, 1994.

ADDRESSES: Comments are to be submitted in quadruplicate or 1 original (hardcopy) and 1 disk (5¼ or 3½) in WP 5.0, 5.1, 6.0 or Ascii to: The Docket Office, Docket No. H-122, Room N-2625, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210, Telephone No. (202) 219-7894. (Any information not contained on disk, e.g., studies, articles, etc., must be submitted in quadruplicate.)

Notices of intention to appear and testimony and evidence are to be submitted in quadruplicate to: Mr. Tom Hall, Division of Consumer Affairs, Occupational Safety and Health Administration, 200 Constitution Avenue, NW., room N3649, Washington, DC 20210; (202) 219-8615.

The hearing will be held in the auditorium of the U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC.

FOR FURTHER INFORMATION CONTACT: *Proposal:* Mr. James F. Foster, Director of Information and Consumer Affairs, Occupational Safety and Health Administration, 200 Constitution Avenue, NW., room N3641, Washington, DC 20210; (202) 219-8151.

Informal Hearing Information: Mr. Tom Hall, Division of Consumer Affairs, Occupational Safety and Health Administration, 200 Constitution Avenue, NW., room N3649, Washington, DC 20210; (202) 219-8615.

Table of Contents

I. Supplementary Information
A. Events Leading to This Action
II. Health Effects
A. Sick Building Syndrome
B. Building-Related Illness
1. Indoor Air Contaminants
2. Microbial Contaminants
C. Environmental Tobacco Smoke
1. Pharmacokinetics
(a) Absorption and Distribution
(b) Metabolism
2. Irritation
3. Pulmonary Effects
4. Cardiovascular Effects
(a) Thrombus Formation
(b) Vascular Wall Injury
(c) Possible Mechanisms of Effect
(d) Acute Heart Effects
(e) Chronic Heart Effects
5. Reproductive Effects
6. Cancer
(a) Evidence of Association
(b) Epidemiological and Experimental Studies
7. Genotoxicity
8. Conclusions
D. Case Reports
1. Sick Building Syndrome and Building-Related Illness
2. Environmental Tobacco Smoke
III. Exposure
A. Sources of Indoor Air Contaminants
B. Microbial Contamination
C. Exposure Studies
1. Low-level Contaminants
2. Bioaerosols
3. Environmental Tobacco Smoke
(a) Chemistry
(b) Human Activity Pattern Studies Used to Assess Workplace Exposure
(c) Indoor Levels of Environmental Tobacco Smoke Constituents
(d) Levels of Respirable Suspended Particulates and Nicotine Found in Field Studies
(e) Biomarkers of Environmental Tobacco Smoke Exposure
(f) Inadequacy of General Dilution Ventilation to Address Environmental Tobacco Smoke Exposure Control
IV. Preliminary Quantitative Risk Assessment
A. Introduction
B. Review of Epidemiologic Studies and Published Risk Estimates
C. Data Sources
D. OSHA's Estimates of Risk-Environmental Tobacco Smoke Exposure
E. OSHA's Risk Estimates—Indoor Air Quality
F. Pharmacokinetic Modeling of Environmental Tobacco Smoke Exposure
1. Considerations for Selection of a Biomarker for Environmental Tobacco Smoke
2. Cardiovascular Effects
3. Carcinogenicity
4. Evaluation of Cotinine as a Biomarker for Environmental Tobacco Smoke
5. Description of Pharmacokinetic Models for Nicotine and Cotinine
6. Application of Pharmacokinetic Modeling for Environmental Tobacco Smoke Exposure Estimation
7. Analysis of Uncertainty
(a) Physiological Parameters
(b) Distribution Parameters
(c) Kinetic Parameters
V. Significance of Risk

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host factors, such as age and immune system status. Mitigation of building-related illnesses requires identification and removal of the source, especially in cases involving hypersensitivity responses.

1. Indoor Air Contaminants

Comments submitted to the docket in response to the RFI and contained in the literature indicate that specific substances or classes of substances have been implicated as contributing to poor indoor air quality problems. These substances, either alone or in synergy, have produced health effects that OSHA believes can be considered material impairment [Ex. 4–124]. In most cases, people likely to be at risk have specific susceptibility.

But such susceptibility is common and adverse effects can arise suddenly following exposure. The relevant effects can be categorized into six categories: irritation, pulmonary, cardiovascular, nervous system, reproductive, and cancer.

Common chemical sense or irritation perception is mediated through receptors found not only throughout the nasal, pharyngeal, and laryngeal areas of the respiratory system but also on the surface of the eyes, specifically the conjunctiva and cornea [Ex. 4–239]. It is partially through the stimulation of these receptors that exposed persons perceive irritation. Many comments to the docket, from citizens, researchers, and indoor air consultants, raised the issue about the irritating effects related to known indoor air contaminants. The air contaminants of concern include formaldehyde [Exs. 3–14, 3–32, 3–38, 3–188, 3–440a, 3–446, 3–575, 4–125, 4–144, 4–214], volatile organic compounds (VOCs) [Exs. 3–32, 3–446, 3–500, 4–145, 4–243, 4–320], ozone [Exs. 3–14, 4–42, 4–134, 4–236, 4–237], carpet-associated chemicals [Exs. 3–25, 3–444D, 3–576, 4–144, 4–214], vehicle exhausts [Exs. 3–6, 3–63, 3–206, 3–238, 3–360, 3–437, 3–444D, 3–631, 3–659], combustion gases [Ex. 3–32], particulates [Exs. 3–32, 3–446, 3–500], man-made mineral fibers (fiberglass, glasswool and rockwool) [Ex. 4–33], and pesticides [Ex. 3–446]. The irritation effects present as sensory irritation of the skin and upper airways, irritation of eye, nose and throat, dry mucous membranes, erythema, headache, and abnormal taste [Ex. 3–14, 4–33]. The pulmonary effects include upper and lower respiratory tract effects such as rapid breathing, fatigue, increased infection rate, broncho-constriction, pulmonary edema, asthma, allergies and flu-like symptoms. Acute exposure to low level of air contaminants results in primarily reversible effects, while chronic exposure may result in pulmonary fibrosis that can result in irreversible damage [Exs. 3–14, 4–33].

These health effects were associated, as reported in many comments to the docket, with specific contaminants, including asbestos [Exs. 3–38, 3–440A, 3–500], combustion gases [Exs. 3–14, 3–34, 3–440A, 3–446, 3–500], formaldehyde [Exs. 3–32, 3–38, 3–188, 3–440A, 4–124], ozone [Exs. 4–42, 4–237], VOCs [Ex. 3–32], vehicular exhaust [Ex. 3–63], and particulates [Exs. 3–32, 3–38, 3–440A, 3–500].

Individuals with underlying pulmonary disease, such as asthma, are more susceptible than others to acute exposure to these indoor air contaminants and experience coughing and wheezing at low levels of exposure. Synergism may occur between chemical contaminants, such as ozone and VOCs, in aggravating asthma [Ex. 4–33]. These affected individuals may also be at increased risk of pulmonary infections due to the synergistic effect between chemical and microbial contaminants [Ex. 4–33].

Cardiovascular effects have also been associated with poor indoor air quality. These effects are presented as headache, fatigue, dizziness, aggravation of existing cardiovascular disease, and damage to the heart. These effects are associated with exposure to combustion gases such as carbon monoxide [Exs. 3–38, 3–440A], VOCs [Ex. 3–500], and particulates [Ex. 3–500].

Nervous system effects have also been produced due to exposure to poor indoor air quality. These effects include headache, blurred vision, fatigue, malaise with nausea, ringing in the ears, impaired judgement, and polyneuritis. These effects are associated with exposure to carbon dioxide [Ex. 3–14], carbon monoxide [Exs. 3–32, 3–38, 3–446, 3–500], formaldehyde [Exs. 3–32, 3–38, 3–446, 3–500], and VOCs [Exs. 3–32, 3–446, 3–500].

Relevant reproductive effects include menstrual irregularities and birth defects and are associated with exposure to formaldehyde [Exs. 3–446, 3–500] and VOCs [Exs. 3–446, 3–500].

The occurrence of cancer has also been attributed to exposures associated with poor indoor air quality. In particular, cancer of the lung, including mesothelioma, esophagus, stomach, and colon have been associated with exposure to asbestos [Exs. 3–6, 3–14, 3–38, 3–188, 3–440A, 3–500], radon [Exs. 3–35, 3–38, 3–188, 3–440A, 3–500], vehicular exhausts [Exs. 3–84, 3–206, 3–360H], combustion gases [Ex. 3–500], VOCs [Exs. 3–446, 3–500, 4–294], and particulates [Ex. 3–500].

2. Microbial Contamination

Building-related illnesses can result in serious illness and death. Indoor transmission of disease caused by obligate pathogens (microbes that require a living host) is common in indoor environments, especially those that are overcrowded and inadequately ventilated [Ex. 4–33]. Diseases in this category include influenza, rhinovirus or colds, and measles. Indoor transmission of disease caused by opportunistic microorganisms usually affects compromised individuals, those with existing conditions that make them more susceptible to infection, such as pulmonary disease or immunodeficiency. Legionnaires' disease, pulmonary tract infections, and humidifier fever are diseases that fall into this category. Diseases that affect the immune system include allergic reactions, as seen in antibody-mediated responses (asthma and rhinitis) and interstitial lung disease, as seen in cell-mediated reactions (hypersensitivity pneumonitis) [Ex. 4–33]. All of these diseases produce substantial amounts of illness each year [Exs. 4–33, 4–41, 4–214].

In the U.S., Legionnaires' disease is considered to be a fairly common, serious form of pneumonia. The *Legionella* bacterium is one of the top three bacterial agents in the U.S. which causes sporadic community-acquired pneumonia. Because of the difficulty in clinically distinguishing this disease from other forms of pneumonia, many cases go unreported. Although approximately 1,000 cases are reported to the Centers for Disease Control and Prevention annually, it has been estimated that over 25,000 cases of the illness actually occur. This disease burden is estimated to result in over 5,000 to 7,000 deaths per year [Ex. 4–41]. Brooks et al. [Ex. 4–33] reported that as many as 116,000 cases occur each year. Of these cases, it is estimated that between 35,000 and 40,000 die. The attack rate for *L. pneumophila* ranges from 0.1 to 5%. The case fatality rate ranges from 15 to 20% [Ex. 4–214].

Two serious allergic or hypersensitivity diseases are asthma and hypersensitivity pneumonitis (extrinsic allergic alveolitis). An estimated 3% of the U.S. population suffers from asthma (approximately 9,000,000 people) [Ex. 4–41]. These individuals may be more susceptible to bioaerosol contamination or chemical contamination of the indoor environment.

Hypersensitivity pneumonitis is triggered by recurrent exposure to microbials, fumes, vapors, and dusts

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Styrene
Urethane
Vinyl chloride
1,1-dimethylhydrazine
2-nitropropane
2-napthylamine
4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone
4-aminobiphenyl
5-methylchrysene
7H-dibenzo(c,g)carbazole
Indeno (1,2,3,-cd)pryene

(b) Epidemiological and Experimental Studies. There are at least 32 epidemiological studies that have attempted to evaluate the carcinogenic potential of ETS. OSHA analyzed these studies and determined that 14 were positive for an association [Exs. 4–36, 4–65, 4–106, 4–119, 4–121, 4–142, 4–143, 4–153, 4–158, 4–187, 4–252, 4–275, 4–276, 4–292, 4–300], 5 were equivocal with a positive trend [Exs. 4–4, 4–47, 4–117, 4–122, 4–171], and 13 were equivocal [Exs. 4–35, 4–38, 4–52, 4–118, 4–148, 4–164, 4–175, 4–183, 4–192, 4–283, 4–286, 4–296, 4–326]. [See the Risk Assessment section for further discussion.]

OSHA considered the consistency of the association to determine if the finding of the same exposure effect occurred in different populations and different types of studies. The great number of epidemiological studies available on ETS were conducted by different researchers, on different populations, in various countries with diverse study designs. This extensive amount of data increases confidence that the associations seen between ETS exposure and the development of lung cancer is externally consistent and is not due to artifacts or a product of some unidentified, indirect factors unlikely to be common to all of the studies. The fact that exposure to ETS is common dilutes the risk estimates derived from these studies because the comparison group has some exposure to ETS. A recent Centers for Disease Control and Prevention (CDC) report [Ex. 4–50] found that 100% of a subset of the National Health and Nutrition Evaluation Survey (NHANES) III conducted by the National Center for Health Statistics had detectable levels of cotinine in their bodies indicating that everyone in the sample had detectable exposure to tobacco smoke [Ex. 4–50]. Cotinine is a metabolite of nicotine and is used as a surrogate of exposure to tobacco smoke. This indicates that the cancer risk may indeed be greater since the relationship in these studies has been more exposed versus less exposed instead of exposed versus nonexposed.

Many potential sources of bias, such as publication bias (the tendency of scientific journals to publish studies with positive results), misclassification bias (smokers or former smokers claiming to be nonsmokers), and recall bias (the reliance on self-reporting of both personal smoking habits and exposure to others' tobacco smoke) can not account for the elevation in risks seen in these various studies. Also, the relative risks that were estimated from prospective study data are similar to those estimated from case/control study data. Biases that may be problematic to case-control studies are not a problem in prospective studies. Since the results from both types of studies are similar it is apparent that these biases are not important in the case-control studies (e.g., misclassification bias and recall bias). This information strengthens the confidence of a causal connection.

Animal studies have shown the carcinogenicity of cigarette smoke. Limited existing data suggest that sidestream smoke may contain more carcinogenic activity per milligram of cigarette smoke concentrate than does mainstream smoke [Ex. 3–689D]. Currently, OSHA is aware of only a few experimental inhalation studies with sidestream smoke or ETS reported in the literature. A discussion of these studies follows.

Otto and Elmenhorst [Ex. 4–247] have shown that there are carcinogenic constituents in the vapor phase of tobacco smoke. They exposed C57B1 and BLH mice to the gas phase of cigarette mainstream smoke of 12 cigarettes for 90 minutes daily over 27 months. The particulate matter was removed by passing the smoke through a Cambridge filter. The percentages of mice with lung adenomas were 5.5% and 32% in the smoke-exposed C57B1 and BLH mice, as compared to 3.4% and 22% for their respective controls. Leuchtenberger and Leuchtenberger [Ex. 4–197] have also shown that the rate of tumors among mice exposed to the gas phase was greater than animals exposed to the whole smoke. Pulmonary adenomas and adenocarcinomas were induced in Snell's mice by the gas phase but not by the whole smoke in this study. These studies demonstrate that the carcinogenicity of tobacco smoke is not limited to the particulate phase.

Studies have also reported hyperplasia and metaplasia in the trachea and bronchi of mice exposed to cigarette smoke by the inhalation route [Exs. 4–226, 4–327]. Four lung tumors and emphysema were detected in 100 male and female C57B1 mice exposed, nose only, to fresh mainstream smoke [Ex. 4–135].

Pulmonary squamous neoplasms were detected in female Wistar rats exposed to a 1:5 smoke-to-air mixture for 15 seconds of every minute during an 11 minute exposure twice a day, 5 days per week, for the lifespan of the animals [Ex. 4–79]. Respiratory tumors were also observed in Fischer–344 rats exposed, nose only, to a 1:10 smoke to air mixture for approximately 30 seconds every minute, 7 hours per day, 5 days per week for 128 weeks [Ex. 4–77]. The incidence of laryngeal leukoplakias in Syrian golden hamsters ranged from 11.3% for the animals that received the low dose to 30.6% of those animals that received the highest dose. These animals were exposed to a 1:7 smoke-to-air mixture for 10 to 30 minutes, 5 days a week, nose only, for a period of up to 52 weeks [Ex. 4–88]. Exposing hamsters twice a day, 5 days a week for up to 100 weeks resulted in almost 90% of the exposed hamsters having hyperplastic or neoplastic changes in the larynx in a study by Bernfeld et al. [Ex. 4–30]. Lung tumors have been reported in beagle dogs exposed to the smoke from nonfilter cigarettes [Ex. 4–19]. However, no tumors were seen in rabbits exposed to cigarette smoke for up to 5½ years [Ex. 4–149].

Sidestream condensates have also been shown to cause carcinogenicity when implanted into female Osborne-Mendel rat lungs [Ex. 4–127]. Cigarette smoke condensate fraction from sidestream smoke was implanted at a dose level of one cigarette per animal in this study.

Coggins et al. [Ex. 4–59] reported epithelial hyperplasia in the nasal cavity of high-dosed rats exposed to environmental tobacco smoke. They exposed Sprague-Dawley rats of both sexes, nose only, to "aged and diluted sidestream smoke" (ADSS) at 0.1, 1 or 10 mg of particulates per meter for 14 days and found "slight to mild" epithelial hyperplasia and inflammation in the most rostral part of the nasal cavity in the 10 mg group only. They also found that these changes were reversible if the animals were kept without further exposure for an additional 14 days. No effects in the lung were reported. Similar results of mild hyperplasia were also obtained when male rats were exposed to the same concentrations for up to 13 weeks [Ex. 4–60]. In this study the authors reported hypercellularity and the thickening of the respiratory epithelium of the dorsal nasal conchae and adjacent wall of the middle meatus.

Rats are obligatory nose-breathers, and the anatomy and physiology of the respiratory tract and the biochemistry of the lung differ between rodents and humans. Because of these distinctions, laboratory animals and humans are

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test. However, no elevation in chromosomal aberrations was observed in alveolar macrophages in this study. Similar results were also obtained when animals were exposed to the same three concentrations for up to 90 days. DNA adducts were seen in lung, heart and larynx DNA of the animals exposed to the highest concentration of ADSS [Ex. 4–195]. The adduct levels were highest after 90 days of exposure and were significantly reduced in all target tissues 90 days after cessation of exposure. Again, chromosomal aberrations in alveolar macrophages were not elevated in any group after 90 days of exposure. The authors concluded that the concentration of DNA adducts formed in the lung tissue did not increase linearly as the ADSS concentration was increased from 1 to 10 mg.

Several short-term tests have been performed in eukaryotic systems. A solution of the gas phase of mainstream cigarette smoke has been shown to induce reciprocal mitotic recombination in *Saccharomyces cerevisiae* D3 and petite mutants in an isolate of strain D3 [Ex. 4–163]. Whole mainstream cigarette smoke induced mitotic gene conversion, reverse mutation, and reciprocal mitotic recombination in strain D7 of *Saccharomyces cerevisiae* [Ex. 4–113]. Transformation of mammalian cells was induced in several cell systems using the cigarette smoke condensate from mainstream cigarette smoke [Exs. 4–22, 4–161, 4–188, 4–267, 4–268, 4–298].

Another *in vitro* assay that measures the number of sister-chromatid exchanges (SCEs) induced has been employed widely to determine the mutagenic activity of cigarette smoke. Valadand-Berrieu and Izard [Ex. 4–313] used a solution of the gas phase from cigarette mainstream smoke and showed that this solution induced a significant dose-related increase in sister-chromatid exchanges. Putman et al. [Ex. 4–257] have also demonstrated dose-dependent increases in sister chromatid exchange frequencies in bone-marrow cells of mice exposed to cigarette smoke for 2 weeks.

Review of the literature clearly demonstrates that MS smoke and ETS exposure causes cancer in humans. These results are supported not only by animal studies but also by studies that show SS smoke to be both genotoxic and clastogenic.

8. Conclusions

The epidemiological and clinical studies, taken in aggregate, indicate that exposure to environmental tobacco smoke may produce mucous membrane irritation, pulmonary, cardiovascular, reproductive, and carcinogenic effects in nonsmokers. Exposure to ETS may aggravate existing pulmonary or cardiovascular disease in nonsmokers. In addition, animal studies show that both mainstream and sidestream tobacco smoke produce similar adverse effects.

D. Case Reports

1. Sick Building Syndrome and Building-Related Illness

Many case reports of material impairment of health due to occupational exposure to poor IAQ have been reported to OSHA through submission to the indoor air quality docket [H–122]. These adverse health effects range from irritation effects to more severe, life-threatening building-related illnesses, such as Legionnaire's disease, and cancer.

Ford Motor Company responded in docket comment 3–447, that "[p]resently, at Ford, we investigate an average of two IAQ complaints per month which are predominantly classified as Sick Building Syndrome. We have seen Building-Related Illness, but these incidents have been rare and associated with specific contaminant episodes. The IAQ complaints we generally investigate are characterized by general malaise, headache, and flu-like symptoms that are said to disappear when the occupants leave the building * * * Of the IAQ problems investigated, about 20 percent can be attributed to PTS [passive tobacco smoke]/ETS. Upper respiratory irritation or eye irritation typically are associated with these complaints." Similar types of health effects were reported to the agency in docket comments 3–1, 3–22, 3–58, 3–142C, 3–367, 3–413, 3–529, 3–632, 3–634, 3–642, 3–659, and 3–698.

One comment [Ex. 3–433 reported that "based upon approximately 30 IAQ investigations in a member company over the past two and one-half years, the following adverse health effects have been reported in office environments: eye, nose, and throat irritations; headaches, nausea, dizziness, fatigue; cough, shortness of breath, chest tightness. These so-called "sick building syndrome (SBS)" symptoms often disappear when the person leaves the building environment. These symptoms are usually subjective and non-specific, lacking a physician's diagnosis of a definite illness." Others have reported [Ex. 3–377] that "as air flow and ventilation are cut back, our workers are becoming sick. Many are exposed to contaminants or other harmful substances; and, without ventilation, these sources linger and cause nausea, skin irritations and other unhealthy symptoms of illness. In severe cases, these contaminants and bacteria have been known to contribute to upper respiratory infections." Comment 3–570 reported similar health effects due to poor indoor air quality.

More serious health conditions have been reported ranging from severe asthma to central nervous systems disorders. For example, Comment 3–158 responded that "I have developed a serious asthma condition due to indoor air quality problems. Besides, three of the remaining five employees at the branch office have been diagnosed with chronic fatigue syndrome. In conversations with various health care professionals, I have come to the conclusion that the diagnoses of chronic fatigue syndrome were actually sick building syndrome. Of the six employees at the branch office, four of the six are moderate to heavy smokers. This does not take into consideration the other factors that could be causing poor indoor air quality problems in the office."

Comment 3–631 was a collection of reports from the workers in one building that illustrate the poor conditions of a building that can lead to serious health effects in workers. Health problems experienced by workers in this building included chronic sinus infections; headaches; fatigue; eye, nose and throat irritations; difficulty breathing and congestion; allergies; and asthma. These health problems seem to clear up when the workers were out of the building over a weekend or a vacation.

The physical condition of this building was obviously in disrepair since the commenters reported pails of stagnant water, collected from leaks in the roof, were left in hallways. Water in "[t]hese pails ha[d] overflowed and run down the stairs. What [wa]s left in the pails evaporate[d] leaving a gross residue of who knows what." The water leaks from the roof caused mold infestation and water damage. Water logged insulation hung in the ceiling out in a hallway. There was an obvious lack of routine, sufficient cleaning. Dust and particulate matter were visible in the air. The bathrooms were dirty. Smells of sewer gas, mold, and diesel and other vehicular fumes permeated the office space. Ventilation problems were evident since paint or varnish fumes lingered whenever part of the inside physical structure of the building was painted. Tar fumes were evident from constant patching of the leaky roof. Insect infestation of the building was evident. Pesticide fumes lingered whenever the building was spray[ed] for roaches and steam bugs. Workers sighted cockroaches, silverfish, and

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TABLE II–1.—URINARY EXCRETION OF NICOTINE AND METABOLITES IN MALE AND FEMALE RATS AFTER INTRAVENOUS ADMINISTRATION OF [^{14}C]NICOTINE (0.5 mg/kg)—Continued

Metabolite	Male		Female	
	Recovery of administered radioactivity (percentage)	$t_{1/2\beta}$ (Hr)	Recovery of administered radioactivity (percentage)	$t_{1/2\beta}$ (Hr)
Nornicotine	8.1 ± 0.9	4.1 ± 0.6	8.1 ± 1.8	[1]8.3 ±1.3
Demethylcotinine	0.8 ± 0.1	ND	<0.3	ND
Γ-(3-Pyridyl)-Γ-oxo-N-Methylbutramide	1.8 ± 0.3	3.5 ± 0.6	[1]0.6 ± 0.3	ND
Isomethylnicotinium ion	2.1 ±	4.5 ± 0.7	<0.3	ND
Allohydroxydemethylcotinine	2.8 ± 0.4	9.8 ± 1.4	1.9 ± 0.6	10.0 ±1.6
Total	69.4 ± 3.0		65.0 ± 3.6	

[1] $0.01 < p \leq 0.05$.
[2] $p \leq 0.01$.
[3] ND, not determined; concentration too low to estimate $t_{1/2\beta}$ accurately.

The ability of tobacco smoke to elicit irritation may be enhanced by low relative humidity and varies according to concentration [Ex. 4–239]. Irritating components of ETS are contained in both the vapor phase and the particulate phase (see Tables III–6 and III–7). These effects have been studied in both experimental (e.g., animals studies; clinical and chamber studies on humans) and field (e.g., surveys and epidemiological studies) studies. The NRC report [Ex. 4–239] summarized these studies and concluded that even though the specific components of ETS that cause irritation were not identified, the overall effects were eye and throat irritation and immunological responses. Weber [Ex. 4–317] reported the results of a field study that included 44 workrooms where smoking was taking place. Eye irritation was reported by 52 out of 167 workers. Nonsmokers reacted more than smokers to the ETS; 36 of the 52 workers who reported eye irritation at work were nonsmokers [Ex. 4–317]. Asano et al. [Ex. 4–18] reported significant eye irritation, as measured by blinking rates, in both healthy smoking and nonsmoking adults following exposure to ETS. Nonsmokers reported more eye irritation than smokers did. Effects such as eye irritation and nasal stuffiness were reported to OSHA in comments to the docket [Exs. 3–38, 3–58, 3–59, 3–188, 3–438D, 3–440A].

3. Pulmonary Effects

Much of the literature relevant to the association between non-cancerous health effects and ETS has focused on children. Because children are undergoing development and maturation, they are not physiologically equivalent to adults exposed to the same conditions. Therefore, findings in studies conducted with respect to ETS and children may not be directly applicable to adults. However, a number of studies have investigated the relationship between ETS and pulmonary health effects in adults.

Studies which are restricted to adults vary by numerous factors, such as the population studied, the measures used to estimate exposure to ETS, and the physiologic and health outcomes examined. The studies also varied in the consideration of potential confounders. A number of studies have found relationships between ETS exposure and pulmonary health effects. These studies have: (1) used pulmonary function tests, which may be more sensitive than methods used in other studies, to detect physiological changes occurring in the small airways of the lungs (e.g., forced mid-expiratory flow rate (FEF_{25-75}), and forced end-expiratory flow rate (FEF_{75-85})); (2) studied older populations with a longer history of exposure to ETS; (3) stratified the level of ETS exposure with significant findings more likely to occur in persons with higher exposures; and (4) more frequently found significant changes in lung function in men, although adverse pulmonary effects to ETS have also been shown in women. The following discussion summarizes the results of these studies [Exs. 4–18, 4–37, 4–62, 4–148, 4–173, 4–176, 4–178, 4–180, 4–209, 4–210, 4–278, 4–295, 4–321].

Asano et al. [Ex. 4–18] demonstrated the acute physiologic changes which occur as a result of exposure to ETS. Nonsmokers had more pronounced changes in eye blinking rates (a measure of eye irritation), expired carbon monoxide, increased heart rate and systolic blood pressure.

Studies of ETS and chronic health effects in adults differ by how they define "never smokers", "exsmokers", and how other various levels of ETS exposure are defined, either in nominal, ordinal or interval scales; and whether or not they take into account exposure both in the workplace and at home. The potential for misclassification bias occurs when "nonsmokers" are loosely defined and used as the comparative group to passive smokers. Several studies considered the confounding impact of environmental air pollution [Ex. 4–278], indoor cooking fuels [Exs. 4–37, 4–62] or occupational exposures to dusts and fumes [Exs. 4–176, 4–178, 4–209, 4–210, 4–321].

There have been fewer longitudinal studies [Exs. 4–148, 4–278, 4–295] as compared to the majority which have been cross-sectional studies. The duration of exposure, which is critical to producing a measurable health effect, was quantified by number of years directly in several studies [Exs. 4–37, 4–148, 4–173, 4–295, 4–321], or indirectly by the age of the population under study [Exs. 4–176, 4–209, 4–210]. In those studies which had carefully assessed for level of exposure and had specified a duration of at least 10 years, significant pulmonary function decrements were noted in both men and women [Exs. 4–37, 4–148, 4 176, 4–321]. Overall, changes in pulmonary indices are more likely to occur in men than in women, however, several studies have documented statistically significant physiological changes in pulmonary function occurring in women [Exs. 4–37, 4–176, 4–178, 4–321].

Understanding the significance of findings is complicated because studies used a variety of measures from spirometry. Although most studies evaluated FVC (forced vital capacity) and FEV_1 (forced expiratory volume in one second), fewer studies have measured FEF_{25-75} or FEF_{75-85} [Exs. 4–176, 4–180, 4–209; 4–210, 4–321]. These later measures have been suggested as being more sensitive to detecting changes in the small airways where effects of ETS are most likely to occur

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1. Pharmacokinetics

Whether a chemical elicits toxicity or not depends not only on its inherent potency and site specificity but also on how the human system can metabolize and excrete that particular chemical. To produce health effects, the constituents of ETS must be absorbed and must be present in appropriate concentration at the sites of action. After absorption, some of these contaminants are metabolized to less toxic metabolites while some carcinogens are activated by metabolism in the body. Available biomarkers of ETS, such as nicotine, clearly show that nonsmoker exposure is of sufficient magnitude to be absorbed and to result in measurable levels of these biomarkers. There is sufficient evidence in the literature to indicate that several components of sidestream smoke are rapidly absorbed and widely distributed within the body. However, the extent of absorption, distribution, retention and metabolism of these contaminants in the body depends upon various physiological and pharmacokinetic parameters that are influenced by gender, race, age and smoking habits of the exposed individuals. These parameters and others may result in differences in susceptibility among exposed subpopulations. Nicotine is one of the most widely studied constituents of tobacco smoke. There have been numerous studies on the pharmacokinetics of nicotine in both animals and man.

(a) Absorption and distribution. Absorption and distribution of tobacco smoke constituents are usually measured by using surrogate markers. A correlation between nicotine absorption and exposure to tobacco smoke has between demonstrated, thus making nicotine an appropriate marker for tobacco smoke in pharmacokinetic studies. The steady state volume of distribution for nicotine is large indicating that it is widely distributed within the body [Ex. 4–185]. Nicotine has been shown to bind with plasma proteins which may interfere with elimination and thereby prolong retention in the body. The studies in the docket clearly indicate that nicotine and other constituents of tobacco smoke are readily absorbed and distributed throughout the body thereby increasing the potential of producing adverse effects at more then one target site.

(b) Metabolism. Nicotine is rapidly eliminated, primarily via metabolism and urinary excretion. The investigation of metabolism *in vivo* and *in vitro*, has resulted in the identification of more than 20 metabolic products in the plasma and urine of humans and animals. The principle metabolic pathways of nicotine appear to involve oxidation of the pyrrolidine ring to yield nicotine-1'-N-oxide and cotinine, the latter being the major metabolite and the precursor of many of the metabolic products of nicotine. Some of the metabolites detected in the urine of rats after intravenous administration in a study by Kyerematen et al. [Ex. 4–185] are listed in Table II–1. In humans, cotinine is the major degradation product of nicotine metabolism and has a serum half-life of about 17 hours compared to two hours for the parent compound, nicotine [Exs. 4–27, 4–253]. Trans-3'-hydroxycotinine in the free form constitutes the largest single metabolite in smokers' urine accounting for 35–40% of the urinary nicotine metabolite [Exs. 4–48, 4–241].

Smokers and nonsmokers differ in their metabolism of nicotine and cotinine [Exs. 4–133, 4–184, 4–279]. The half-life values for urinary elimination of nicotine and cotinine were found to be significantly shorter in smokers than nonsmokers [Ex. 4–186]. Plasma nicotine clearance was faster in smokers than in nonsmokers in this study. More rapid elimination of nicotine and cotinine has been attributed to the inductive effects of chronic cigarette smoking on the hepatic metabolism of many xenobiotic agents. However, Benowitz et al. [Ex. 4–29] were unable to confirm published research suggesting that smokers metabolize nicotine and cotinine more rapidly than nonsmokers.

Variations in nicotine metabolism occur among individuals. Variations also occur due to differences in gender and race [Exs. 4–26, 4–186, 4–314]. It has also been suggested that the metabolism of nicotine between smokers and nonsmokers may differ. Male smokers have been shown to metabolize nicotine faster than do female smokers after intravenous infusion of nicotine and active smoking. However, this difference was not observed by Benowitz and Jacob [Ex. 4–23] during a study of daily intake of nicotine in smokers versus nonsmokers. The metabolism of nicotine has also been studied in animals. Male rats (4 strains) were shown to metabolize nicotine faster than did females [Ex. 4–185].

In summary, the potential effect of nicotine, and other ETS constituents in the body, is governed by interactions between several physiological and pharmacokinetics parameters. These interactions may lead to longer retention of toxic constituents, thus prolonging the effects on the target organs resulting in tissue injury.

2. Irritation

Exposure to ETS is capable of inducing eye and upper respiratory tract irritation. Common chemical sense or irritation perception is mediated through receptors in the fifth, ninth, and tenth cranial nerves. These receptors are found throughout the nasal, pharyngeal, and laryngeal areas of the respiratory system and also on the surface of the eyes [Ex. 4–239]. It is partially through the stimulation of these receptors that exposed persons perceive irritation.

TABLE II–1.—URINARY EXCRETION OF NICOTINE AND METABOLITES IN MALE AND FEMALE RATS AFTER INTRAVENOUS ADMINISTRATION OF [^{14}C]NICOTINE (0.5 mg/kg)

Metabolite	Male		Female	
	Recovery of administered radioactivity (percentage)	$t_{1/2\beta}$ (Hr)	Recovery of administered radioactivity (percentage)	$t_{1/2\beta}$ (Hr)
Nicotine	10.8 ± 1.5	2.5 ± 0.4	[1]24.0 ± 4.6	[2]5.6 ± 0.5
Cotinine	9.3 ± 0.8	6.0 ± 0.6	[1]5.7 ± 0.7	[2]6.8 ± 0.8
Nicotine-N-oxide	10.8 ± 0.9	1.6 ± 1.4	7.8 ± 1.4	2.6 ± 0.3
Cotinine-N-oxide	8.5 ± 1.6	7.5 ± 0.8	[1]3.7 ± 1.0	6.8 ± 0.6
3-Pyridylacetic acid	1.8 ± 0.3	5.8 ± 0.3	1.2 ± 0.2	[3]ND
Γ3-(3-Pyridyl)-Γ-oxobutyric acid	2.7 ± 0.6	5.3 ± 0.9	2.4 ± 0.7	6.0 ± 0.6
3-Hydroxycotinine	5.7 ± 0.5	6.7 ± 0.8	5.6 ± 1.5	9.9 ±1.5
Γ-(3-Pyridyl)-Γ-methylaminobutyric acid	4.2 ± 0.6	5.9 ± 0.8	[1]1.4 ± 0.4	ND

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indoor air pollution was first objectively demonstrated in 1984 in a study of 62 Danish subjects suffering from "indoor climate symptoms" [Ex. 4–20]. These subjects reported primarily eye and upper respiratory irritation, but were otherwise healthy individuals, and did not suffer from asthma, allergy, or bronchitis. The subjects were exposed to a mixture of 22 volatile organic chemicals commonly found in the indoor environment at concentrations of 0, 5, and 25 mg/m³. These concentrations corresponded respectively to "clean" air, average polluted air in Danish houses, and maximum polluted air in Danish houses. After exposure, the Digit Span test was administered. The Digit Span test consists of the subject being allowed to view a series of random digits for a short period of time; the numbers are then covered up and the subject asked to repeat the sequence backwards. This test is reported to be sensitive to situational anxiety and alertness, and therefore a measure of stress and ability to concentrate. Bach et al. found significant declines in performance on the digit span test following exposure to these low levels of volatile organic chemicals, demonstrating objectively the existence of SBS [Ex. 4–20].

Molhave et al. [Ex. 4–228], in reporting on the same 62 subjects, found that subjects exposed for 2¾ hrs did not adapt, and that the subjects reacted to irritation of the mucous membranes and not to odor intensity. The exposure was doubled-blind, and neither the subjects nor the testers knew the exposure.

Although these problems have been demonstrated to be real, they may affect only a small percentage of building occupants. Also, there are various degrees of problems which may occur. Some individuals who experience relatively mild and treatable symptoms such as headache, may be able to cope with the sick building environment for extended periods, although suffering from increased stress. Other individuals, more seriously affected, may find symptoms so severe that they may be unable to be in the building for extended periods, or at all. Still others may become temporarily or permanently disabled.

It has been suggested that SBS may not be one syndrome but a number of sub-syndromes [Ex. 4–170]. This hypothesis suggests that the symptoms particularly associated with chemical exposure include fatigue; headache; dry and irritated eyes, nose, and throat; and sometimes include nausea and dizziness. Those symptoms most related to microbial exposures would result in itchy, congested, or runny nose; itchy watery eyes; and sometimes include wheezing, tight chest, or flu-like symptoms. The overlapping symptoms in each case are eye, nose, and throat irritation, perhaps making the two sub-syndromes, chemical and microbial, difficult to distinguish. Jones concludes that there is a need for a treatment protocol as well as a diagnostic protocol, which, in addition to describing corrective actions available in response to different diagnostic findings, would also provide guidelines for the design and implementation of follow-up studies of buildings and individuals in order to assess treatment effectiveness [Ex. 3–170].

Randolph and Moss [Ex. 4–258] have written about a number of problems ascribed to indoor air pollution in the chemically sensitive patient. These problems include irritability from natural gas fumes, allergy to dust from forced air ventilation systems, intoxication and even hallucination from paint fumes. Randolph describes chemical sensitivity to dry cleaning chemicals, and rug shampoo, and implicates moldy carpets in producing allergenic substances. He also describes joint pain, malaise, and fatigue due to pesticide exposure; and skin rashes from exposure to plasticizers. Randolph further describes intolerance to highly scented products such as deodorant soaps, toilet deodorants, and disinfectants, especially pine-scented ones. Other patients have reported reacting to strong perfumes and other cosmetics. So-called air fresheners often prove to be particularly troublesome. He also describes that some patients are sensitive to the odors from hot plastic-coated wires in electronic equipment.

There is little data on the perceptions of victims of SBS. Shapiro [Ex. 4–282] has complied a summary of 16 case-histories of SBS in the victims' own words. It is useful to review these for insight into the problems from the victims' point of view.

One episode that Shapiro [Ex. 4–282] reported on was in a building occupied by a government agency. As a result of problems related to carpeting and other suspected causes, five workers were reported to have left the agency, 11 were relocated to alternative workspace or worked at home, and 100 reported to the agency's medical officer that they had SBS related problems. The range of self-reported symptoms included a variety of moderate and acute respiratory problems; headache; sore throat; burning of the eyes, lungs, and skin; rashes; fatigue; laryngitis; clumsiness; disorientation; loss of balance; nausea; numbness in extremities and face; and difficulty with mental tasks.

The patient's reported that the diagnoses of the occupational health physicians they visited included upper and lower respiratory irritation, intoxication-type syndrome, occupational asthma, and chronic hypersensitivity pneumonitis.

The central nervous system effects reported by many do not lend themselves to ready diagnosis [Ex. 4–282]. Some of the lesser affected individuals either saw no physician at all or saw a family doctor or allergist who was not familiar with occupational or environmental health [Ex. 4–282].

The Air Force Procedural Guide [Ex. 4–199] on dealing with SBS takes a practical view: "* * * in most cases the sick building syndrome does not have a clearly understood etiology and many of the SBS studies and investigations were inconclusive. The significance of exposure that [what chemical or physical agent concentrations cause symptoms] can be pathogenic remains unanswered, but the realities of worker complaints and discomfort are valid reasons to seriously address this problem."

In summary, SBS is not a well-defined disease with well-defined causes. It appears to be a reaction, at least in part due to stimulation of the common chemical sense, to a variety of chemical, physical or biological stimuli. Its victims display all or some of a pattern of irritation of the mucous membranes, and the worst affected individuals have neurological symptoms as well.

B. Building-Related Illness

Building-related illness (BRI) describes specific medical conditions of known etiology which can often be documented by physical signs and laboratory findings. Such illnesses include sensory irritation when caused by known agents, respiratory allergies, nosocomial infections, humidifier fever, hypersensitivity pneumonitis, Legionnaires' disease, and the symptoms and signs characteristic of exposure to chemical or biologic substances such as carbon monoxide, formaldehyde, pesticides, endotoxins, or mycotoxins [Exs. 3–61, 4–144]. Some of these conditions are caused by exposure to bioaerosols containing whole or parts of viruses, fungi, bacteria, or protozoans. These illnesses are often potentially severe and, in contrast to SBS complaints, are often traceable to a specific contaminant source, such as mold infestation and/or microbial growth in cooling towers, air handling systems, and water-damaged furnishings. Symptoms may or may not disappear when the employee leaves the building. Susceptibility is influenced by

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[Ex. 4–33]. The lung interstitium, terminal bronchioles, and alveoli react in an inflammatory process that can organize into granulomas and progress to fibrosis. The symptoms of acute episodes of this disease are malaise, fever, chills, cough and dyspnea. The symptoms of chronic episodes are serious respiratory symptoms such as progressive dyspnea. Chronic disease can lead to irreversible pulmonary structural and functional changes [Ex. 4–33].

Approximately 15% (20,250) of 135,000 hospital admissions per year that last an average of more than eight days are due to allergic disease [Ex. 4–41]. Burge and Hodgson estimate that these hospitalizations cost five million work days per year. The prevalence of symptoms consistent with hypersensitivity pneumonitis, an interstitial lung disease caused by organic dusts or by aerosols has been examined in subpopulations at well-defined, increased risk, such as farmers (0.1–32%) or pigeon breeders (0.1–21%) [Exs. 4–41, 4–214]. The only unbiased source of complaint rates in unselected office workers are control buildings used in the study of hypersensitivity pneumonitis in the U.S. Arnow et al. [Ex. 4–15] reported complaints consistent with hypersensitivity pneumonitis in 1.2 percent and Gamble et al. [Ex. 4–116] in 4 percent of these populations. Since no clinical data are available, it is not known how these complaints are related to actual disease, and it is unknown whether these complaints are associated with lost work time, doctor visits or hospital admissions [Ex. 4–41].

Humidifier fever, a less serious variant of hypersensitivity pneumonitis, also is caused by exposure to microorganisms contained in an aerosol. Attack rates in building epidemics have been as high as 75%, whereas complaint rates are usually 2–3% in nonepidemic situations [Ex. 4–41]. Because of the similarity of the individual symptoms to other diseases (fever, headache, polyuria, weight loss and joint pain), it is often difficult to separate actual disease from complaints related to the common cold in nonepidemic situations [Exs. 4–33, 4–41]. While rare, a workplace epidemic of humidifier fever can virtually shut down an entire building, and only removal of the contamination will end the epidemic [Exs. 4–41, 4–144, 4–214].

Microbial contamination of building structures, furnishings, and HVAC system components contribute to poor indoor air quality problems, especially those related to building-related illnesses. OSHA believes that consequent health effects constitute material impairment of health [Exs. 3–61, 4–41]. These can be categorized as irritation, pulmonary, cardiovascular, nervous system, reproductive, and cancer effects.

Irritation effects, either from the physical presence of bioaerosols or from exposure to VOCs released by biologicals, have been demonstrated in susceptible workers [Ex. 3–32]. In addition, water leakage on furnishings or within building components can result in the proliferation of microorganisms that can release acutely irritating substances into the air. Typically, where microorganisms are allowed to grow, a moldy smell develops. This moldy smell is often associated with microbial contamination and is a result of VOCs released during microbial growth on environmental substrates [Ex. 4–41].

Pulmonary effects which have been associated with exposure to bioaerosols include rhinitis, asthma, allergies, hypersensitivity diseases, humidifier fever, spread of infections including colds, viruses, and tuberculosis, and the occurrence of Legionnaire's disease [Exs. 3–17, 3–32, 3–38, 3–61B, 3–188, 3–440A, 3–446, 3–500, 4–41, 4–144, 4–214].

Building-related asthma has also recently been documented in office workers [Exs. 3–61, 4–43] and some case reports show it to be associated specifically with humidifier use. Biocides used in humidification systems are suspected causes of office-associated asthma [Ex. 4–103].

Cardiovascular effects manifested as chest pain, and nervous system effects manifested as headache, blurred vision, and impaired judgment, have occurred in susceptible people following exposure to bioaerosols [Exs. 3–32, 3–446]. It has been suggested that these effects may be caused by VOCs released by the microbiologicals, or they may be a complication of related pulmonary effects.

The development of cancer in susceptible people is possible following exposure to certain types of toxigenic fungi and mycotoxins. However, the probability of such exposures occurring in workplaces covered by this standard is probably limited. Mycotoxins (toxins produced as secondary metabolites by many fungi) are among the most carcinogenic of known substances, and are also acutely toxic. The American Conference of Governmental and Industrial Hygienists wrote "[t]he toxigenic fungi are common contaminants of stored grain and other food products and have caused well-described outbreaks of acute systemic toxicosis as well as specific organ carcinogenesis when such food is consumed * * * It appears clear that massive contamination with a highly toxigenic fungus strain of a site in which aerial dispersion of metabolic products occurred would be necessary to induce acute symptoms. However, considering the carcinogenicity of many fungal toxins, an examination of the risks of chronic inhalation exposure appears justified" [Ex. 3–61].

In summary, most of the health effects associated with SBS and BRI occur in indoor environments were concentrations of pollutants are much less than the OSHA Permissible Exposure Levels (PELs) (29 CFR 1910.1000) [Ex. 4–3]. It is important to point out that the PELs are chemical-specific standards that are not only based on health effects but also on technological feasibility, cost restraints and a "healthy" worker exposed for a 40-hour work week. In the industrial workplace, hazards are minimized by the use of administrative and engineering controls and the use of personal protective equipment. The nonindustrial environment, however, does not have these controls. Ventilation systems are designed only to remove occupant-generated contaminants, such as carbon dioxide and odors. These types of systems were not designed to dilute multiple point sources of contaminants that are typically found in nonindustrial workplaces (see section III). Unless adequate ventilation and source controls are utilized and adequately maintained, many of the chemical contaminants can concentrate to levels that induce symptoms. The possibility exists that synergistic effects occur. These effects occur not only between substances to enhance their toxicity but also by lowering the resistance to lung infection in susceptible persons.

C. Environmental Tobacco Smoke

ETS is composed of exhaled mainstream and sidestream smoke. The chemical composition and exposure sources of ETS are described in the Exposure section of this preamble (see Section III). The pharmacokinetics of ETS have been widely studied and are described in the following section.

A wide spectrum of health effects have been associated with exposure to ETS. These effects include mucous membrane irritation, decrease in respiratory system performance, adverse effects on the cardiovascular system, reproductive effects, and cancer. The following section also presents more detailed information on these health effects.

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compared to home (10%). When the data set was examined by the presence or absence of smokers in the home, however, subjects who lived with smokers had virtually equivalent exposures across all three settings: work (34%), home (36%), and "other" (31%). Nonsmokers living with smokers received 29 minutes per day of exposure at work and 31 minutes per day at home and 27 minutes per day in other settings. On the other hand, subjects who did not live with smokers had the majority of their exposure at work (36 minutes per day) and very little at other settings.

Additional studies verify that the workplace is an important source of exposure to ETS, particularly for nonsmokers unexposed at home [Exs. 4–172, 4–262, 4–315]. A U.K. study of exposure to ETS in 20 nonsmoking men whose wives smoked showed that 78% of the men's reported hours of exposure came from outside the home; by contrast, 90% of the ETS exposure of 101 nonsmoking men whose wives did not smoke was reported to come from non-domestic microenvironments [Ex. 4–315]. Repace and Lowrey [Ex. 4–262] estimated that 86% of the U.S. population was exposed to ETS, and that the workplace was more important than the home as a source of ETS exposure, when weighted by the duration, exposure intensity, and probability of exposure. Kabat and Wynder [Ex. 4–172], in a study of 215 sixty-year-old U.S. women nonsmokers, found that 65% reported exposure to ETS at home and 67% reported exposure at work, averaged over adulthood.

The conclusion that can be made from the activity surveys is that the workplace is a major location of ETS-exposure to nonsmokers. Human activities that involve contact with a greater number of people increase the probability of contact with smokers, and thus with ETS. These studies indicate that the workplace, with its high person densities relative to other microenvironments, including the home, appears to be a major factor in the working nonsmoking population's ETS exposure.

(c) Indoor Levels of Environmental Tobacco Smoke Constituents. Personal monitoring studies have confirmed the role of the workplace as an important microenvironment of ETS exposure to nonsmokers. Spengler et al. [Ex. 4–288] and Sexton et al. [Ex. 4–280] demonstrated by personal monitoring of respirable suspended particulates (RSP) and the use of time-activity questionnaires that exposures to ETS both at home and at work are significant contributors to personal RSP exposures. Coultas et al. [Ex. 4–66], in a pilot study of 15 nonsmokers in Albuquerque, New Mexico, collected questionnaires and samples of saliva and urine to determine workplace ETS exposure. Personal air samples were obtained pre- and post-workshift. Exposure to ETS was reported by 13 of the 15 subjects. The mean number of hours of exposure was 3.4 (±2.1). Basically, although the levels of cotinine, respirable particles, and nicotine varied with self-reports of ETS exposure, the general trend was a direct relationship between increasing incidence of self-reporting of exposure and actual biomarker data. Coghlin, Hammond, and Gann [Ex. 4–61] found similar results for 53 nonsmoking volunteers studied by use of personal nicotine monitors, diaries, and questionnaires. They also found that the closer a nonsmoker was to a smoker, the higher the probability that the nonsmoker would report exposure.

Presently, vapor phase nicotine and respirable suspended particulate matter (ETS–RSP) are the most commonly used markers for ETS because of their ease of measurement, knowledge of their emission rate from tobacco combustion, and their relationship to other ETS contaminants [Ex. 4–311]. Controlled experiments have shown that vapor phase nicotine varies with the source strength, and shows little variation among brands of cigarettes. Field studies have also shown that vapor phase nicotine concentrations are correlated with the number of cigarettes smoked, and further that weekly average nicotine concentrations are correlated with ETS-RSP [Ex. 4–311].

(d) Levels of Respirable Suspended Particulates and Nicotine Found in Field Studies. Respirable suspended particulates (RSP) and nicotine are the most commonly used surrogates for ETS exposure [Ex. 4–239]. Both chamber and field studies have demonstrated that tobacco combustion has a major impact on indoor RSP mass when particle size is under 2.5 microns [Ex. 4–239]. A few examples illustrating the impact of ETS on nicotine and RSP concentrations in workplace and domestic microenvironments are shown in Tables III–10 and III–11. Studies of RSP in public access buildings by Leaderer et al. [Ex. 4–190], First [Ex. 4–105], and Repace and Lowrey [Exs. 4–260, 4–261] (a total of 42 smoking buildings and 21 nonsmoking buildings) showed that the weighted average RSP level during smoking in the smoking buildings was 262 $\mu g/m^3$, while in the nonsmoking buildings the RSP level average 36 $\mu g/m^3$.

Leaderer and Hammond [Ex. 4–189] measured weekly average vapor phase nicotine and RSP concentrations in 96 residences. Vapor phase nicotine measurements were found to be closely related to number of cigarettes smoked and highly predictive of RSP generated by tobacco combustion. The mean RSP background in the absence of measurable nicotine was found to be 15.2±7 $\mu g/m^3$. The mean RSP value in the presence of nicotine was 44.1±30 $\mu g/m^3$. The weekly mean nicotine concentration in the 47 residences with detectable nicotine values was 2.17 $\mu g/m^3$ (Table III–10).

Summary statistics of additional studies on personal monitoring for nicotine are shown in Table III–11 [Ex. 4–263]. These studies show that the median exposures ranged from 5 to 20 $\mu g/m^3$.

Summary nicotine data analyzed by the U.S. EPA [Ex. 4–311] suggest that average nicotine values in residences where smoking is occurring will average 2 to approximately 10 $\mu g/m^3$, with peak values of 0.1 to 14 $\mu g/m^3$ as shown in Table III–10. Offices with smoking occupants show a range of average nicotine concentrations similar to that of residences, but with considerably higher peak values. RSP mass concentrations in smoker-occupied residences show average increases of from 18 to 95 $\mu g/m^3$, with individual increases as high as 560 $\mu g/m^3$ or as low as 5 $\mu g/m^3$. ETS–RSP concentrations in offices with smoking occupants on average appear to be about the same as in residences. Restaurants, transportation, and other indoor spaces with smoking occupants have a generally wider range of increases in particle mass concentrations due to ETS than residential or office environments [Ex. 4–311].

In summary, field data show that RSP is elevated by one to two orders of magnitude during smoking, and that nicotine released during smoking is easily detectable in both homes and workplaces by area or personal monitors. Offices with smoking occupants show a range of average nicotine concentrations similar to that of residences (2 to 10 $\mu g/m^3$), but with considerably higher maximum values. ETS–RSP concentrations in offices with smoking occupants on average appear to be about the same as residences (18 to 95 $\mu g/m^3$). Restaurants, transportation, and other indoor spaces with smoking occupants have a generally wider range of particle mass concentrations due to ETS than residential or office environments [Ex. 4–311]. It must be noted that measurements of nicotine and ETS–RSP in indoor spaces do not

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constitute a direct measure of total exposure. Concentrations measured in all microenvironments have to be combined with human activity pattern studies to determine the time-weighted sum of various exposures.

(e) Biomarkers of Environmental Tobacco Smoke Exposure. Nicotine, and its metabolite, cotinine, and other tobacco smoke constituents in the saliva, blood and urine have been used as biomarkers of active and passive smoking. Nicotine and cotinine can be used to determine the integrated short-term exposure of ETS across all microenvironments [Ex. 4–311].

TABLE III–10.—MEAN NICOTINE LEVELS IN HOME AND WORKPLACE AIR: AREA MONITORS [1]

Study and location	Sample	µg/m³	Comment
Leaderer and Hammond 1991, homes, NY State	47	2.17	7-day average smoking.
Hammond [3–1096] Mass., industrial	.....	24	9-hour average workshift (non-smoker's air; smoking allowed on premises).
White collar	60	21.5	
Blue collar	123	8.9	
Food service	51	10.3	
Carson (1988), offices, Canada	31	11	Workday samples.
Miesner (1989) workplaces, MA	11	6.6	Workweek average.
Oldaker (1990), restaurants, NC	33	10.5	1-hour average (range).
Jenkins (1991), Knoxville, TN, metro	.....	.....	≥1-hour average.
Restaurants	7	3.4	
Cocktail lounges	8	17.6	
Bowling alleys	4	10.7	
Gaming parlors	2	10.7	
Laundromats	3	2.0	
Airport gates	2	6.0	
Office	1	6.0	
Nagda (1989), U.S. aircraft—in-flight average:			
All flights	69	13.4	Smoking section.
Domestic	61	0.11	Nonsmoking section.
International	8	0.33	Nonsmoking section.
Vaughn (1990), highrise office building	1	2.0	Nonsmoking air; 9-hour average.

[1] Adapted from Repace and Lowrey 1993 [Ex. 4–263].

Table III–11.—NICOTINE IN NONSMOKERS' AIR: PERSONAL MOITORS [1]

Study and Location	Sample	µg/m³	Comment
Schenker (1990), railroad clerks, NE	40	6.9	Workshift median.
Coultas (1990), white collar, NM	15	20.4	Workshift mean ± SD.
Mattson (1989); flight attendants	4	4.7	4 flights, mean ± SD.

[1] Adapted from Repace and Lowrey 1993 [Ex. 4–263].

Both nicotine and cotinine are tobacco-specific. Cotinine in saliva, blood, and urine is the most widely accepted biomarker for integrated exposure to both active smoking and ETS by virtue of its longer half-life than nicotine in body fluids. The half-life of cotinine in nonsmokers is of the order of a day, making it a good indicator of integrated ETS exposure over the previous day or two [Ex. 4–311]. Although intersubject variability exists for both nicotine absorption and cotinine metabolism [Exs. 4–156, 4–162], cotinine is a good indicator that ETS exposure has taken place [Ex. 4–311]. Further, studies show that cotinine levels correlate with levels of recent ETS exposure [Ex. 4–311].

In summary, nonsmokers' exposure to ETS has been characterized by a database of widely used atmospheric and biological markers which have been measured in a number of workplaces, such as offices, restaurants, commercial buildings, and on trains and in planes. OSHA believes that this database is sufficient to support the risk assessment which follows. ETS-nicotine exposures of the average worker appear to be of the order of 5 to 10 micrograms per cubic meter (µg/m³), and for the most-exposed workers, 50 to 100 µg/m³. For ETS-RSP, exposures are about tenfold that of the nicotine levels. The concentrations of various ETS atmospheric markers to which nonsmokers are exposed in the workplace, such as nicotine, respirable suspended particulate matter (RSP) and carbon monoxide, are linearly correlated with the amount of tobacco burned. Studies of human activity patterns show that the workplace is the largest single contributor to ETS exposure. Air exchange rates in nonindustrial workplaces are not designed to control the risks of ETS exposure.

(f) Inadequacy of General Dilution Ventilation to Address Environmental Tobacco Smoke Exposure Control. A primary function of heating, ventilating, and air-conditioning (HVAC) systems is to circulate air throughout a building to achieve thermal and sensory comfort for the building occupants. The general ventilation function of the HVAC system is to dilute and remove occupant generated bioeffluents and other contaminants from the space. However, from the industrial hygiene perspective, general ventilation as delivered by a HVAC system, is not an acceptable engineering control measure for controlling occupational exposures to ETS.

Dilution ventilation offers no protection in those cases where, due to the close proximity to a smoker (e.g., contaminant point source), the nonsmoking employee may be exposed to large amounts of sidestream smoke and exhaled mainstream smoke (ETS). Due to the limitations of general ventilation, the smoke cannot be removed from the air before reaching

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[Exs. 4–46, 4–216, 4–230, 4–231]. However, there is no clear consensus in the medical literature as to the routine clinical use of FEF_{25-75} or FEF_{75-85}, or their diagnostic value in independently detecting small airway disease [Ex. 4–8].

Estimates of the decrement in FEV_1 due to ETS exposure in passive smokers as compared to never smokers, ranges from 80 milliliters (ml) [Ex. 4–148] to 190 ml [Ex. 4–37]. When this decrement is expressed as a percent of FEV_1, it has been estimated to be 5.7% in males, or 7.3% when these same subjects were matched for age [Ex. 4–210]. As a means of comparison, the average loss in lung volume per year due to aging alone is estimated to be 25 to 30 ml [Ex. 4–329]. The American Thoracic Society [Ex. 4–8] specifies that spirometry equipment have a level of accuracy within 50 ml. Since pulmonary function maneuvers are very effort dependent, intra-individual variation between the three best efforts should be within 5% to be acceptable. The importance of these spirometry criteria is emphasized by the fact that the FEV_1 may result in being 100 to 200 ml lower than when a maximal effort is given by the subject. Furthermore, a decrease of 15% must be achieved before certain pulmonary indices are considered outside of normal limits. Given this perspective, although changes in pulmonary function tests may truly occur as a result of exposure to ETS over a number of years, the actual clinical impact may not be apparent in the healthy, young individual. Older individuals and those with preexisting pulmonary disease are more susceptible to the pulmonary effects of exposure to ETS.

Outside of respiratory changes being documented through pulmonary function testing, other symptoms have been found to be significantly associated with ETS exposure. Hole et al. [Ex. 4–148] found a significant increase in the prevalence of infected sputum, persistent sputum, dyspnea and hypersecretion in passive smokers as compared to controls. Furthermore, rates increased as those exposed were stratified by level of exposure to passive smoke from low to high. Kauffmann et al. [Ex. 4–178] noted a significant increased risk for dyspnea in American (Odds Ratio (OR)=1.42) and French women (OR=1.43), and an increased risk for wheeze in American women (OR=1.36). Schwartz and Zeger [Ex. 4–278] found an increased risk for phlegm or sputum in a 3-year longitudinal study (OR=1.41). This risk was raised to 1.76 when asthmatics, who may be medicated, were excluded from the analysis.

As small airway disease progresses to chronic obstructive pulmonary disease (COPD) (also referred to as chronic obstructive lung disease (COLD)), the impact of ETS becomes more detectable. Kalandidi et al. [Ex. 4–173] reported an adjusted odds ratio of 2.5 (90% Confidence Interval (CI), 1.3 to 5.0) for Greek women never smokers exposed to their husbands' tobacco smoke.

While there is a clear trend, and in several studies a statistically significant finding of a demonstrated decrease in pulmonary function indices, or an increase in respiratory symptoms in passive smokers, the impairment nonsmokers suffer by the exposure may not be immediately obvious. It is important to note that these findings have been demonstrated in otherwise healthy individuals. Based upon the finding of White and Froeb [Ex. 4–321], Fielding and Phenow [Ex. 4–102] have described such changes as being equivalent to those found in light smokers, who smoke from 1 to 10 cigarettes per day. Where a decrease of 100 to 200 ml of FVC or FEV_1 may be clinically insignificant in healthy persons, such a change may be significant for workers with already impaired pulmonary function [Exs. 3–438D, 3–440A, 4–76, 4–182]. These changes may be the pivotal point at which a worker becomes unable to continue to work.

Cellular effects on the pulmonary tissue have also been observed in animals exposed to ETS during experimental studies. Several studies reviewed by OSHA have demonstrated that chronic cigarette smoke exposure produces an accumulation of alveolar macrophages (AM) (the presence of AM indicates a body's response to environmental insults), within the respiratory bronchioles of many animals species. This effect is similar to that seen in human smokers [Exs. 4–31, 4–58, 4–109, 4–110, 4–140, 4–147, 4–150, 4–179, 4–212, 4–249]. Increased elastase secretion by alveolar macrophages from mice chronically exposed to cigarette smoke has also been observed [Ex. 4–322].

Accumulation of polymorphonuclear leucocytes (PMNs) is also an indication of the body's response to environmental insults. PMNs were found in the alveolar septum of cigarette smoke-exposed hamsters, similar to the PMNs observed in the lungs of human smokers [Ex. 4–204]. In contrast to the focal nature of the alveolar macrophages accumulation, the accumulation of PMN is diffuse. Studies of PMN leukocyte function have not been systematically evaluated in smoke-exposed animals.

Other studies also show effects of ETS exposure at the cellular level. For example, young lambs exposed to ETS for one month did not develop detectable pulmonary system effects or alteration in lung mechanics or airway responsiveness. However, the lambs did develop inflammation of pulmonary cells [Ex. 4–290]. A cytotoxic effect of tobacco smoke was also demonstrated by decreased intracellular adenosine triphosphate (ATP) content in guinea pig alveolar macrophages and lowered cell bacteriocidal activity in a study by Firlik [Ex. 4–104]).

Exposure to tobacco smoke has been shown to increase the permeability of the respiratory epithelial membrane to macromolecules. Burns et al. [Ex. 4–45] have shown that exposure of guinea pigs to tobacco smoke followed by fluorescein isothiocyanate-dextran (FITC–D, molecular weight 10,000) increased the amount of intact FITC–D that crossed the respiratory epithelium into the vascular space. Transmission electron-microscopic studies showed that the FITC–D diffused across damaged type I pneumocyte membranes and cytoplasm to reach the basal lamina and entered the alveolar capillaries through the endothelial junction. Damage to alveolar epithelium was more frequent for the smoke-exposed animals than the room air-exposed animals.

Aryl hydrocarbon hydroxylase (AHH) participates in the activation of various carcinogens, such as benzo(a)pyrene. This is one of the many carcinogens found in ETS. Both mainstream and sidestream smoke are capable of inducing pulmonary AHH activity. Gairola [Ex. 114] has demonstrated the induction of pulmonary AHH activity in Sprague-Dawley rats and male C57BL mice after exposure to either mainstream or sidestream smoke from University of Kentucky Reference cigarettes (2R1) for seven days per week for 16 weeks. However, no such induction was noted in Hartley guinea-pigs under similar conditions, indicating a species difference. The mainstream and the sidestream smoke were equally effective in inducing the AHH activity.

There is consistent evidence that decrements in pulmonary function and increases in respiratory symptoms occur in current smokers and in exsmokers. However, in passive smokers these health effects are not as easily demonstrated. The Environmental Protection Agency's December 1992 report, Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders [Ex. 4–311], reviewed an abundance of evidence showing

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Helsing study, which was done in the 60's—a time when smoking was more acceptable than more recently, and being a prospective cohort study, was less prone to misclassification and other sources of bias. The Fontham study used multiple sources to ascertain nonsmoking status and validate subject response. Study subjects were questioned twice; the self-reported nonsmoking status was corroborated by urinary cotinine measurements; and medical records were cross-referenced with the physician's assessment. In addition, in the Fontham study, information on occupational exposure was collected and an estimate of lung cancer risk attributable to the workplace exposure was ascertained.

The annual risk of disease attributable to occupational exposure to ETS was estimated by dividing the expected number of cases (N_e) by the number of nonsmoking workers in the U.S. population. Table IV–10 presents the annual risk attributable to occupational exposure to ETS per 1,000 exposed employees. Because section (6)(b)(5) of the OSH Act states that no employee shall suffer "material impairment of health or functional capacity even if such an employee has regular exposure to the hazard dealt with * * * for the period of his working life", OSHA has converted the attributable annual risk into an attributable lifetime risk on the assumption that a worker is employed in his or her occupation for 45 years. Lifetime estimates of risk attributable to occupational ETS are presented in Table IV–10. Information contained in Table IV–10 indicates that for every 1,000 workers exposed to ETS, approximately 1 will most likely develop lung cancer and 7 to 16 will develop heart disease if they are exposed to ETS at their workplace in the course of a 45-year working lifetime. The formula used to calculate lifetime risk estimates the probability of at least one occurrence of disease in 45 years of continuous exposure and assumes independence of events from year to year. It also assumes that the worker's exposure profile and working conditions that may affect the level and intensity of exposure remain constant throughout a working lifetime.

TABLE IV–10.—ESTIMATES OF RISK FOR NONSMOKING WORKERS EXPOSED TO ETS AT THE WORKPLACE [1,2]

	Annual risk [2]	Lifetime occupational risk [3]
Lung cancer	0.01–0.02	0.4–1

TABLE IV–10.—ESTIMATES OF RISK FOR NONSMOKING WORKERS EXPOSED TO ETS AT THE WORKPLACE [1,2]—Continued

	Annual risk [2]	Lifetime occupational risk [3]
Heart disease ...	0.15–0.36	7–16

[1] Risks are expressed as number of cases per 1,000 workers at risk.

[2] The annual risk for nonsmoking workers is estimated assuming the proportion of nonsmoking workers exposed to ETS at the workplace ranges from 18.81 to 48.67.

[3] Assumes 45 years of occupational exposure and is calculated as $1-(1-p)^{45}$, where p is the annual risk.

E. OSHA's Risk Estimates—Indoor Air Quality

Adverse health effects associated with poor IAQ are described as Building-Related Illness (BRI) and Sick Building Syndrome (SBS). SBS related conditions are not easily traced to a single specific substance, but are perceived as resulting from some unidentified contaminant or combination of contaminants. Symptoms are relieved when the employee leaves the building and may be reduced by modifying the ventilation system.

Research in Britain [Ex. 4–44], Denmark [Ex. 4–284] and the United States [Ex. 3–745] indicates that about 20% of all office workers are afflicted with such symptoms. If the 20% level were to be considered as "background", a simple approach would be to determine that any building, more than 20% of whose occupants report the symptoms, would be considered to be "sick". However, the question then arises as to how much greater than 20% would the incidence have to be to be considered excess and how would one address such issues as statistical significance for any one building. Furthermore, the definition used in assessing symptom occurrence can cause substantial variations in estimating symptom prevalence, even in the same building. The problem with many investigations of "sick" buildings is that rarely have "non-sick" or control buildings been used to determine background prevalence of the symptoms. Until now, it appears that limited research has been done to address the issue of background levels of symptoms. OSHA seeks input on data sources to address expected background levels of SBS related conditions.

Mendell and Smith [Ex. 4–218] examined symptom reports compiled in a number of individual studies for a number of buildings which had different types of ventilation. On the basis of the information gathered in the individual studies, Mendell and Smith compared the prevalence of sick building symptoms in buildings with five types of ventilation: natural only; fans only; air conditioned with no humidification; air conditioned with steam humidification; and air conditioned with water-based humidification. Overall, they found the prevalence of work-related headache, lethargy, upper respiratory/mucous membrane, lower respiratory and skin symptoms significantly increased in buildings with any type of air conditioning as compared to buildings with no air conditioning. Thus, according to this analysis, a basic problem with SBS appears to reside in the air conditioning system or, in some building aspect associated with the presence of air-conditioning.

Building-related illness (BRI) describes those specific medical conditions of known etiology which can often be documented by physical signs and laboratory findings. Symptoms may or may not disappear when the employee leaves the building. Currently, OSHA does not have any data on BRI related symptoms to conduct a quantitative risk assessment.

The number of cases of illness in the United States related to poor indoor air quality has not yet been quantified; however OSHA has made an attempt to develop a preliminary risk estimate of SBS using a similar methodology as was done for ETS. The National Health Interview Survey was the primary data source for U.S. population frequency rates for acute upper respiratory symptoms other than the common cold, influenza, acute bronchitis, and pneumonia and frequency rates on severe headaches other than migraines. For this preliminary risk assessment, OSHA used the reported frequency rates as representative of population incidence rates for upper respiratory conditions and severe headaches. OSHA seeks comment on the use of frequency data in place of incidence data.

Observed relative risks for comparable conditions were estimated by Mendell [Ex. 4–219]. Mendell's data source was the California Healthy Building Study. This study surveyed a representative sample of 12 public office buildings in Northern California to ascertain the occurrence of work-related symptoms associated with air-conditioned office buildings. All buildings were either smokefree or had separately ventilated designated smoking areas. The sample included 6 buildings with air-conditioning systems, 3 buildings with mechanical ventilation and no air-conditioning, and 3 buildings with

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report published in December, 1992 by the Environmental Protection Agency, addressing hazards associated with exposure to ETS. In that study, Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders [Ex. 4–311], EPA concluded that exposure to ETS presents an excess risk of induction of cancer in humans. OSHA has submitted this proposed standard to the U.S. Environmental Protection Agency which is reviewing it in detail for purposes of submitting detailed comments to the docket.

For the reasons noted above, and discussed in the following sections, OSHA is proposing to address indoor air quality problems, including exposure to ETS, as set forth in this notice.

II. Health Effects

Indoor air quality problems can occur in all types and ages of buildings; in newly constructed buildings, in renovated or remodeled buildings, and in old buildings. Problems in new, clean buildings are rarely, if ever, related to microbial growth, since the physical structures are new [Ex. 3–61]. Older buildings that have not been adequately maintained and operated may have problems with bioaerosols if parts of the building have been allowed to become reservoirs for microbial growth. Also, if inadequate outside air is provided, regardless of the age of the building, chemical and biological contaminants will build up to levels that can cause health effects in some workers. In addition, other physical factors such as lack of windows, noise, and inadequate lighting, and ergonomic factors involving uncomfortable furniture and intensive use of video display units, etc., will cause discomfort in occupants that may be inaccurately attributed to air quality.

Some information contained in the docket indicates that these chronic health complaints are psychological, however, OSHA believes that chronic health complaints related to poor indoor air quality are unlikely to be due to mass psychogenic illness, even though a psychological overlay is common. It is true that poor management, boring work, poor lighting conditions, temperature variations, poor ergonomic design, and noise may all lower the threshold for complaint. Nevertheless, air quality complaints usually have some basis, although they are often difficult to assess with specificity [Exs. 3–61C, 4–144].

Indoor air quality problems are generally classified as Sick Building Syndrome (SBS) or Building-related Illness (BRI). However, a very important constituent of poor indoor air quality is ETS because of the serious health effects that result from exposure. The following discussion will first identify the health effects associated with SBS and BRI. A discussion of the health effects associated with exposure to ETS will follow.

It is important to note that OSHA considers these health effects to be material impairments of health when the worker is clinically diagnosed with a condition that is either caused or aggravated by poor indoor air quality in the workplace. For example, in the formaldehyde standard [29 CFR 1910.1048] [Ex. 4–107] OSHA determined that a physician's diagnosis of irritation met the requirement of material impairment of health. In addition, OSHA considers all the other health effects discussed, which are more clinically severe than irritation, to be material impairments of health as well.

A. Sick Building Syndrome

Typically, health effects caused by poor indoor air quality have been categorized as SBS or BRI. In 1983, the World Health Organization published a list of eight non-inclusive symptoms that characterize Sick Building Syndrome [Ex. 4–325]. These include irritation of the eyes, nose and throat; dry mucous membranes and skin; erythema; mental fatigue and headache; respiratory infections and cough; hoarseness of voice and wheezing; hypersensitivity reactions; and nausea and dizziness. Generally, these conditions are not easily traced to a specific substance, but are perceived as resulting from some unidentified contaminant or combination of contaminants. Symptoms are relieved when the employee leaves the building and may be reduced or eliminated by modifying the ventilation system. Comments to the docket indicate that such symptoms have been observed in and reported by workers [Exs. 3–446, 4–87].

In some instances, outbreaks of SBS are identified with specific pollutant exposures, but in general only general etiologic factors related to building design, operation and maintenance can be identified [Ex. 4–274]. In 1987, Woods et al. [Ex. 3–745] conducted a stratified random telephone survey of 600 U.S. office workers across the national. Twenty four percent reported that they were dissatisfied with the air quality at the office; while 20% perceived their performance to be hampered by poor indoor air quality. Women were nearly twice as likely to report a productivity effect of poor indoor air quality than men (28% versus 15%). Based on this, Woods et al. [Ex. 3–745] hypothesized that 20% of U.S. office workers are exposed to indoor conditions which manifest as SBS. In fact, complaints about SBS have become so numerous that 37 out of 53 states and territories have designated a building complaints investigation contact person [Ex. 4–310].

Breysse [Ex. 4–32] reported on symptoms associated with new carpeting in a state office building, in order of prevalence: headache, eye and throat irritation, nausea, dizziness, eye tearing, chest tightness, diarrhea, cough, muscle aches, burning nose, fatigue, dark urine, and rashes. Twenty out of 35 persons were affected. Air sampling was conducted before and after carpet removal; a similar range of aliphatic hydrocarbons was found after removal, but in much lower concentrations. Many individuals who believe the building they work in is implicated in SBS, have described similar effects. Symptoms usually include one or more of the following: mucous membrane (eye, nose, or throat) irritation, dry skin, headache, nausea, fatigue, and lethargy [Ex. 4–293]. These symptoms are generally believed to result from indoor air pollution. There is no secondary spread of symptoms to others outside the building who are exposed to the occupants (unlike the situation faced by many chemical and asbestos workers). Anderson [Ex. 4–10] suggested the possible causes for SBS as related to psychosocial, chemical, physical, or biological factors.

Anderson [Ex. 4–10] distinguished SBS symptoms as different from mass psychogenic illness; although in general the causes of SBS are unknown, he suggested that most SBS symptoms could be explained by stimulation of sensory nerve fibers in the upper airways and the face (referred to as common chemical sense). Because these fibers can respond in only one way, SBS cases largely have the same symptoms irrespective of the cause [Ex. 4–10].

It is now known that there is a variety of important health effects from indoor air pollution. In addition to the indoor environmental disease caused by infectious agents, carcinogens or toxins; the indoor environment may create conditions that can produce skin and mucosal allergy and hyperactivity reactions, sensory effects (odors and irritations), airways effects (from both acute and chronic exposures), neuropsychological effects, and psychosocial effects, especially due to the lack of social support [Ex. 4–200].

Indoor air pollution may be caused by physical, chemical, or microbiological agents, and is aggravated by poor ventilation. The causation of SBS by

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on to consumers. In this case, the maximum expected price increase is calculated by dividing the estimated compliance cost for each industry by the sales for that industry. Table VI–13 shows that the average price increase related to the cost of this proposed standard would be extremely small, 0.07 percent, with the largest being 0.41 percent (Personal Services, SIC–72). The results in Table VI–13 indicate that even if all costs were passed on to consumers through price increases, the rule would have a negligible impact on prices.

In many industries, however, establishments will not be able to pass along the entire cost of compliance through price increases since consumers may respond by reducing demand. Such establishments will have to absorb from profit the costs they cannot pass through. If all costs were absorbed from profit, the maximum expected decrease in profit can be calculated by dividing the estimated compliance cost for each industry by its estimated profit. Table VI–13 shows that the average decline in profits under this worst-case-elasticity assumption would be less than 0.94 percent. The largest potential decline in profits would be in Fishing at 4.5 percent (SIC–9).

Because most establishments will not find it necessary to absorb all of the costs from profits and will be able to pass some of the costs on to consumers. average profits will not decline to the extent calculated in this analysis.

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component of one's total CO exposure is to measure workplace CO levels and predict blood HbCO with a physiologically based pharmacokinetic model for CO [Ex. 4–11]. A difficulty associated with the use of CO or HbCO as a biomarker for ETS effects is the presence of other sources of CO in the workplace.

Nicotine can be measured directly in body fluids and the circulating concentration can be related to physiological effects, such as heart rate [Ex. 4–26]. Alternatively, measurements of nicotine in air or cotinine in body fluids can be measured, and the circulating concentration of nicotine can be inferred using a pharmacokinetic model. The use of a pharmacokinetic model to relate inhaled nicotine to circulating nicotine and cotinine levels is the main focus of this section.

PAHs are inhaled in the form of particulates on which they are adsorbed. Developing an appropriate biomarker for ETS-associated PAHs is complicated by the presence of PAHs on particulates not associated with ETS, and by the low, and variable, composition of PAHs adsorbed to particulate matter. One candidate material which has been suggested as an environmental marker for ETS-associated particulates is solanesol, a non-volatile tobacco constituent. However, the pharmacokinetic information necessary for use of solanesol as an internal biomarker is not currently available.

The use of these three different biomarkers (CO, PAHs, and solanesol) does not appear to be practical. It appears that the most effective strategy currently achievable would be to rely on nicotine (or cotinine) measurement as a specific marker of ETS exposure as well as a direct measure of nicotine exposure.

3. Carcinogenicity

The mechanism of carcinogenicity from exposure to ETS is not known, but it has been established that ETS includes a number of chemicals which have been identified as carcinogens (see Tables II–2, III–6, and III–7), although most of the identified carcinogenic components of ETS are not unique to ETS. Therefore, direct measurement of the carcinogenic components or related biomarkers in biological fluids would not provide a unique measure of exposure from ETS. The potentially carcinogenic components of ETS include highly volatile chemicals such as formaldehyde and benzene, lower volatility chemicals such as the nitrosamines, and non-volatile chemicals such as PAHs and metal compounds, which are bound to particulates. Given the current lack of information on the mechanism of carcinogenicity of ETS it is impossible to identify which components of ETS should be targeted for exposure estimation. The most prudent choice for a biomarker in this case would be one which provides the most general representation of all the components of ETS, and which is itself unique to ETS. In an experimental study of potential ETS-unique environmental markers of exposure, only nicotine was found to represent both the gas phase and particulate phase organic constituent of ETS [Ex. 4–97]. Several studies have shown a strong correlation between measurements of nicotine in the air and the mutagenicity of ETS [Exs. 4–198, 4–215]. In these studies, the relationship of nicotine to mutagenicity was as good as or better than the relationship of RSP to mutagenicity (RSP is assumed to be the major contributor of the carcinogenic effects of ETS). Therefore, since measurements of nicotine in the air correlate better than measurements of RSP to mutagenicity of ETS, and there is a positive correlation between short-term mutagenicity tests and carcinogenicity, the use of nicotine as an exposure marker for the carcinogenic effects of ETS appears to be justified.

4. Evaluation of Cotinine as a Biomarker for ETS

The purpose of this section is to discuss the use of cotinine, a metabolite of nicotine, as an internal biomarker for inhalation exposure to nicotine, and, as such, its usefulness as a metric for the health effects of ETS. Cotinine is preferred over nicotine as an internal biomarker because of its slower clearance from the body [Ex. 4–71].

There is a strong correlation between nicotine intake and plasma cotinine levels [Ex. 4–115]. There is also a strong correlation between cotinine measured in body fluids and ETS exposure. In a controlled study, urinary cotinine was found to be a reliable marker for long-term ETS exposure, and plasma and salivary cotinine were found to be good indicators of short- as well as long-term exposure [Ex. 4–73]. Several studies have also demonstrated a positive relationship between self-reported exposure to ETS and cotinine in serum [Exs. 4–166, 4–250, 4–301], saliva [Ex. 4–166], and urine [Exs. 4–166, 4–211, 4–316]. In general, the currently available data support the assumption that nicotine and cotinine kinetics parameters for smokers can be extrapolated to nonsmokers for estimating exposures to ETS in nonsmokers [Ex. 4–24]. Studies have also demonstrated that salivary levels of cotinine are directly proportional to plasma levels [Ex. 4–73], and that urinary excretion of cotinine is linearly related to plasma levels [Ex. 4–82]. Thus all three biological fluids provide a reasonable metric for nicotine intake, and thus can serve as biomarkers of ETS exposure in nonsmokers.

There are two potential difficulties associated with the use of cotinine as a biomarker for ETS. The first is the presence of nicotine in the diet. Several foods, including tea, tomatoes, and potatoes, have been shown to contain nicotine in measurable quantities [Exs. 4–49, 4–81, 4–281]. However, a study of 3,383 nonsmokers was unable to substantiate an effect of tea drinking on serum cotinine levels for self-reported daily tea consumption [Ex. 4–301]. The same study did find a strong correlation between self-reported ETS exposure and serum cotinine level.

OSHA seeks comment and data on whether dietary intake of nicotine should be considered a significant factor in modelling nicotine metabolism for assessing risk due to ETS exposure.

The second issue associated with the use of cotinine as a biomarker is the possibility that there is a longer half-life for the elimination of cotinine at very low biological concentrations, associated with the slow release of nicotine from binding sites [Exs. 4–28, 4–24, 4–167, 4–254]. This longer half-life at very low concentrations could have the effect of overestimating exposure to ETS in the lowest exposed population. At this time there is not sufficient evidence to quantify the potential magnitude of this effect, but it is likely to be small. OSHA seeks comment on this issue.

5. Description of Pharmacokinetic Models for Nicotine and Cotinine

For many purposes, an essentially first order process such as the kinetics of cotinine can be effectively modeled with a simple compartmental kinetic analysis [Exs. 4–27, 4–24, 4–73, 4–82]. The compartmental approach has been used to relate steady-state urinary cotinine levels to atmospheric nicotine concentrations [Ex. 4–263]. For investigating some of the concerns associated with the use of cotinine as a biomarker, however, a physiologically based pharmacokinetic (PBPK) description would be preferred. The advantage of the PBPK approach stems from its biologically motivated structure, which permits the direct incorporation of biochemical data and the biologically constrained comparison of model predictions with experimental timecourses to investigate such issues as dose-rate effects, exposure-route

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Society) [Ex. 4–7]. Data on the U.S. workforce were obtained from the Bureau of Labor Statistics [Ex. 4–39]. Based on the 1993 annual averages, as estimated by the Household Survey, BLS reports that the U.S. workforce for sectors covered by this standard is estimated to be 101,631,300 (men: 54.36%, women: 45.64%). Information on the proportion of employed adults who smoke was obtained from the National Health Interview Survey and is found in Table IV–7 [Ex. 4–235]. It is estimated that 74,201,000 adults (73.01% of the U.S. labor force), employed in sectors covered by this standard, are nonsmokers.

[Editorial note: No Table IV–6 is included in this preamble.]

TABLE IV–7.—PERCENT ESTIMATES OF ADULTS EMPLOYED IN THE UNITED STATES BY SMOKING STATUS[1]

	Smoker	Non-smoker
Currently employed	26.99	73.01
Unemployed	40.38	59.62
Not in labor force	21.50	78.50

[1] National Health Interview Survey [Ex. 235].

In an effort to characterize prevalence of occupational exposure, OSHA considered several sources. To determine the prevalence of smoking among U.S. adults during 1991, the National Health Interview Survey-Health Promotion and Disease Prevention (NHIS–HPDP) supplement collected self-reported information on smoking exposure at work from a representative sample of the U.S. civilian, non-institutionalized population greater than 18 years of age [Ex. 4–51]. In particular, employed individuals were asked whether, during the past two weeks, anyone had smoked in their immediate work area. Based on results adjusted for nonresponse and weighted to reflect national estimates, 18.81 percent of nonsmokers reported exposure to smoke in their immediate work area as shown in Table IV–8. OSHA believes that 18.81 percent may be an underestimate of frequency of exposure in the workplace because it is based solely on self-reported information and the question was not very specific in defining immediate work area.

TABLE IV–8.—PERCENT ESTIMATES OF RESPONSES TO QUESTION 6A IN THE NHIS BY SMOKING STATUS[1]

	Smoker	Non-smoker
Yes	37.58	18.31
No	60.81	79.79
Unknown	1.61	1.39

[1] Question 6a was: "During the past 2 weeks, has anyone smoked in your immediate work area?"

Another source considered by OSHA for defining nonsmoker ETS exposure in the workplace was the work published by Cummings et al. [Ex. 4–67]. A recent re-analysis of the data file showed that among the nonsmoking, currently employed subjects, 48.67 percent (165 out of 339) reported exposure to ETS at work and not at home (Table IV–9) [Ex. 4–69]. Based on the data sources mentioned above, OSHA assumes that the percent of nonsmoking workers who are potentially exposed to ETS at their worksite ranges between 18.81 and 48.67.

TABLE IV–9.—PREVALENCE OF ETS EXPOSURE FOR NONSMOKING WORKERS [1]

Subject category	Count	Percent
Exposed at work and home	99	29.22
Exposed at home, not at work	23	6.78
Exposed at work, not at home	165	48.67
Not exposed at work or home	52	15.34

[1] Data source: Cummings reanalysis [Ex. 4–69].

D. OSHA's Estimates of Risk—Environmental Tobacco Smoke Exposure

The incidence of disease due to occupational exposure in nonsmokers was estimated using the following methodology: The expected number of cases, N_e, in nonsmoking workers who are occupationally exposed to ETS is expressed by:

$$N_e = N_d - N * I_u = N * (I_p - I_u)$$

where:

N_e is the cases in nonsmoking exposed workers attributable to ETS per year

N_d is the estimated number of cases per year in nonsmoking workers

N is the number of nonsmoking workers in the U.S.

I_u is the incidence rate of disease among the unexposed workers

I_p is the U.S. population incidence rate for nonsmokers

The number of nonsmoking workers (N) was estimated by multiplying the percent of currently employed adults who report to be nonsmokers by the number of adult, employed, civilian noninstitutional population, as reported by BLS.

The number of nonsmoking workers with disease per year (N_d) was estimated as $N_d = N * I_p$. The U.S. population incidence rate of lung cancer for nonsmoking women is reported to be 0.121 per one thousand nonsmoking women. The lung cancer incidence for nonsmoking males is estimated to be higher. For the purpose of this risk assessment, OSHA used 0.121 as the population incidence rate of lung cancer for nonsmokers. This will most likely result in an underestimate of the true risk for male workers. The average annual incidence rate for death from coronary heart disease excluding strokes for nonsmokers age 35 to 64 is estimated to be 4 per one thousand men and 2 per one thousand women, as reported by the Framingham study. This results in an overall weighted average of 3 deaths per one thousand individuals.

The incidence rate of disease (I_u) among the unexposed workers is estimated using the relationship:

$$I_u = I_p / [RR * p_e + (1 - p_e)]$$

where:

RR is the observed relative risk of disease for nonsmokers exposed to ETS

p_e is the proportion of nonsmoking workers exposed to ETS while at work.

OSHA used 1.34 as an observed estimate of relative risk (RR) for lung cancer among nonsmokers with occupational exposure as reported by Fontham et al. [Ex. 4–106]. Estimates of observed relative risk for heart disease in nonsmokers, as reported by Helsing et al. (1.24 for females and 1.31 for males), were used in calculating an overall adjusted relative risk estimate of 1.28 [Ex. 4–139]. The adjusted relative risk was a weighted average of the reported relative risks using the gender composition of the U.S. workforce as weights ((1.24*45.64 + 1.31*54.36/100) = 1.28). The proportion of nonsmoking workers exposed to ETS while at work (p_e) was assumed to range from 18.81 to 48.67 as stated previously.

OSHA chose to rely on the Fontham and Helsing studies for estimates of the observed relative risks for several reasons. Both studies were conducted in the U.S. Both are large, population-based studies whose results can be generalized to the general public. Both studies, by design, controlled for misclassification to a large degree. The

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persistent physiologic changes in children's respiratory function and related health effects as a result of exposure to ETS. Studies evaluating these same effects are not as plentiful in adults. However, the EPA concluded, "recent evidence suggests that passive smoking has subtle but statistically significant effects on the respiratory health of adults" [Ex. 4–311].

The weight of the evidence shows that exposure to ETS results in decreases in pulmonary function indices and increases in respiratory symptoms in otherwise healthy men and women who are exposed to ETS for periods of 10 or more years. The risk of developing COPD appears to be increased in passive smokers with lifelong exposures to ETS. Whether these changes impact upon respiratory function to a degree that impairment occurs may be dependent upon the individual's pulmonary status and overall health condition.

4. Cardiovascular Effects

A developing body of research indicates that the cardiovascular effects of ETS exposure on the health of nonsmokers include acute effects, such as exacerbation of angina, as well as chronic effects, such as atherosclerosis [Exs. 4–123, 4–291, 4–330].

Cardiovascular diseases [Exs. 4–91, 4–136] such as myocardial infarction [Ex. 4–12], sudden death, and arterial thrombosis occur more frequently in cigarette smokers as opposed to nonsmokers [Exs. 4–86, 4–233]. The same chemicals which produce these effects in active smokers are present in ETS. These include nicotine, carbon monoxide, polycyclic aromatic hydrocarbons (PAHs) and tobacco glycoproteins.

The following discussion on cardiovascular effects covers thrombus formation, vascular wall injury and the possible mechanisms of these effects in nonsmokers. Discussion of the acute and chronic health effects follows.

(a) Thrombus Formation. Blood clots in the coronary arteries are an important component of an acute myocardial infarction (MI). An additional component of the acute MI is the presence of atherosclerotic plaques in the walls of the coronary arteries. Platelets are involved in both the acute formation of blood clots and the chronic formation of atherosclerotic plaques.

There is evidence that ETS exposure can cause platelets to become more easily activated thus predisposing the platelets to become involved in forming clots and atherosclerotic plaques. For example, evidence exists that demonstrates that the platelets of nonsmokers exposed to ETS are more easily activated [Exs. 4–40, 4–80]. The study by Burghuber [Exs. 4–40] demonstrates that the platelet activating capabilities of ETS are more prominent in nonsmokers than in smokers. The results of this study suggest that nonsmokers are at a greater risk of blood clot formation secondary to ETS exposure than smokers.

Acute ETS exposure also results in an increased platelet aggregation, which is an initial stage of the development of coronary thrombosis or vasoconstriction. This vasoconstriction can lead to the development of coronary atherosclerosis after chronic exposure [Exs. 4–111, 4–123, 4–272]. Environmental smoke exposure also can increase platelet-activating factor (PAF), platelet factor 4, beta-thromboglobulin, and fibrinogen concentration which provides a marker of its effect on coronary heart disease [Exs. 4–85, 4–157, 4–224].

(b) Vascular Wall Injury. Atherosclerotic plaque formation is a complicated chronic process that can lead to constriction of the lumen of the blood vessels, resulting in reduced blood supply to the myocardial tissues. It is thought that an essential step in plaque formation is injury to the endothelial lining of the arterial wall. ETS has been implicated in causing injury to the endothelial cells which line the arterial walls. This was demonstrated in the study by Davis et al. [Ex. 4–80] which identified an increase in the number of endothelial cell carcasses in the circulation of healthy people after being exposed to ETS.

ETS has also been implicated in stimulating smooth muscle cell proliferation and in altering blood lipids. Each of these can contribute to plaque formation which leads to an increased susceptibility to heart attacks.

(c) Possible Mechanisms of Effect. At least three mechanisms are described in the literature by which ETS may place stress on the heart by increasing myocardial oxygen demand, decreasing myocardial oxygen supply or interfering with the cell's ability to utilize oxygen for energy production.

One mechanism by which ETS may reduce oxygen supply is through the formation of carboxyhemoglobin. Carboxyhemoglobin is formed when a person is exposed to carbon monoxide, a component of ETS. The carbon monoxide effectively competes with oxygen for the heme group of the hemoglobin molecule in the red blood cell (RBC). In fact, carbon monoxide has a much greater affinity for hemoglobin than does oxygen and binds very strongly with hemoglobin making it unavailable for the transport of oxygen. The heart muscle (myocardium) can experience injury at the cellular level when the oxygen demanded by the heart muscle exceeds the oxygen supplied by the blood. Therefore, the formation of carboxyhemoglobin can decrease the ability of the blood to deliver oxygen to the myocardium and can cause injury to the heart if myocardial oxygen demand exceeds supply.

A number of studies have suggested that ETS exposure adversely affects the myocardial oxygen supply-demand relationship; this would predispose the heart to develop ischemia or exacerbate preexisting ischemia. Direct or indirect exposure to tobacco smoke has been shown to increase the hemodynamic determinants of myocardial oxygen demand [Exs. 4–13, 4–242] at the same time that it potentially reduces both myocardial oxygen supply and delivery by enhancing the development of coronary atherosclerosis [Exs. 4–242, 4–323], causing coronary vasoconstriction [Exs. 4–323, 4–324] and reducing the oxygen carrying capacity of blood through increased carboxyhemoglobin levels [Ex. 4–13]. As a result, fewer red blood cells are available to transport oxygen to the body, and to the heart muscle itself. To compensate for this reduced oxygen carrying capacity of the blood, the heart must work harder, for example, by increasing the heart rate. This is an example of one mechanism by which ETS may place even further stress on the heart by increasing myocardial oxygen demand, precisely at a time when the oxygen delivery capabilities of the blood are reduced.

A second mechanism by which ETS may increase myocardial oxygen demand is via the direct effect of nicotine. The nicotine in ETS may cause an increased resting heart rate and blood pressure in exposed individuals.

One study examined the effects of ETS on healthy individuals during exercise, and found that healthy individuals experienced fatigue at lower work levels when exercising in the presence of ETS [Ex. 4–123]. The authors concluded that ETS exposure interfered with the heart muscle cells' ability to utilize oxygen for energy production.

Consequently, ETS exposure may have an adverse impact on myocardial metabolism and expose the heart muscle to an increased susceptibility to injury. These mechanisms of cardiac stress and potential injury to the heart are in agreement with accepted theories of cardiac injury.

(d) Acute Heart Effects. An acute effect of exposure to ETS is the aggravation of existing heart conditions.

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changes in control strategies to meet new objectives (e.g., energy conservation) or inadequate design.

Building-related illnesses (BRI), on the other hand, are medically diagnosed diseases that present symptoms that can last for weeks, months, years or even a lifetime. Examples include nosocomial infections, humidifier fever, hypersensitivity pneumonitis, and legionellois. BRI can develop as a result of poor building systems operation and maintenance and uncontrolled point sources of contaminants.

No building has a complete absence of problems, but those that function with minimal occupant complaints and comply with acceptable criteria for occupant exposure, system performance, maintenance procedures and economic objectives may be characterized as healthy buildings. Figure VI–1 below presents the classification of buildings by stages of performance.

Based on the information submitted to the docket, OSHA assumed that 30 percent of the buildings have indoor air quality problems [Ex. 3–745].

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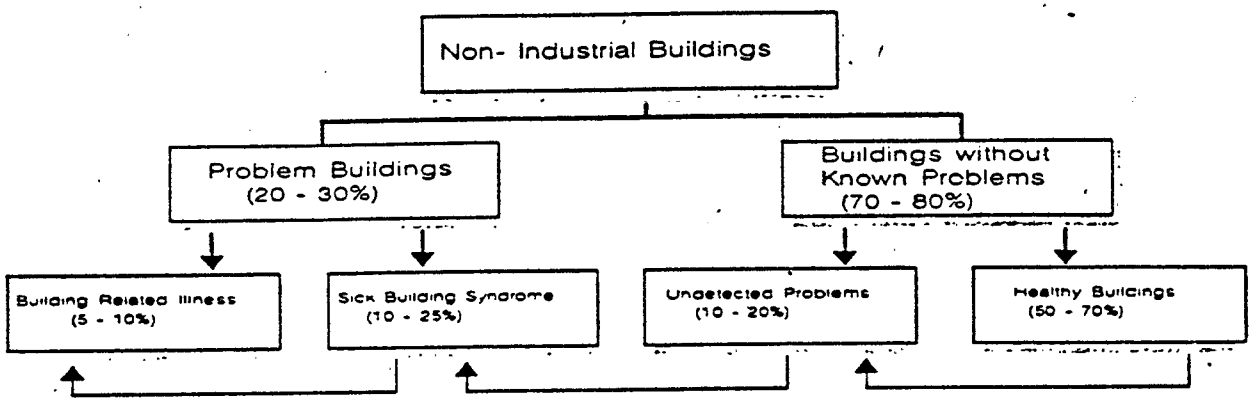
Figure VI-1
Characteristics of Environmental Population of Non-Industrial Buildings



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Therefore, as presented in Table VI–3, the total number of problem buildings is estimated to be 1.4 million buildings.

7. Number of Employees Affected

The commercial building energy consumption survey estimates that there are 70.7 million employees. However, survey data do not provide information by two-digit SIC. OSHA examined data obtained through the Bureau of Labor Statistics to estimate the number of employees by two-digit SIC affected by the proposed standard. The data from the Bureau provided occupational breakdown of the labor force by detailed industry categories (two-digit SIC) and major occupational groupings.

TABLE VI–3.—NUMBER OF PROBLEM BUILDINGS AND NUMBER OF EMPLOYEES EXPOSED TO INDOOR AIR QUALITY PROBLEMS [1]

	Employees working indoors [2]	Number of buildings with IAQ problems	Number of employees exposed to IAQ problems [3]
Agriculture, forestry, fishing	279,050	51,956	83,715
Mining	180,700	4,554	54,210
Construction	1,643,750	128,091	493,125
Manufacturing	5,748,000	77,573	1,724,400
Transportation	3,412,350	48,563	1,023,705
Wholesale and retail trade	15,744,000	384,466	4,723,200
Finance, insurance, real estate	7,248,150	104,863	2,174,445
Services	26,926,000	385,235	8,077,800
Government	9,473,561	173,100	2,842,068
Total	70,655,561	1,358,400	21,196,668

[1] Exclusive of exposure to ETS.
[2] OSHA estimate based upon BLS's 1993 employed persons by detailed industry and major occupation.
[3] Based on OSHA estimate of 30 percent employee exposure to poor IAQ.
Source: OSHA, Office of Regulatory Analysis, 1994.

OSHA classified employees according to whether or not they work primarily in indoor areas, e.g., areas with possible exposures, by developing percentages of employees in each occupational category who might be working indoors. For example, personnel in the transportation industries were apportioned according to those potentially exposed to indoor air pollution (office workers) and those who are not (truck drivers). Table VI–3 presents the distribution of the 70.7 million employees who work indoors.

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such as angina. The National Research Council (1986) reported, based on the effects of studies by Anderson et al. [Ex. 4–9] and Aronow et al. [Exs. 4–14, 4–16, 4–17], that angina patients are especially sensitive at carboxyhemoglobin levels between 2 and 4%. Guerin et al. [Ex. 4–129] report that physiologically adverse effects occur in humans at 2.5% carboxyhemoglobin blood content. Cumulative carbon monoxide levels, due to ETS that result in such an effect are not uncommon in work environments [Ex. 4–129]. Acute exposure to ETS has been reported to increase heart rate, elevate blood pressure, and increase carboxyhemoglobin levels in both angina patients [Exs. 3–38, 4–222] and in healthy subjects [Exs. 4–18, 4–217]. Acute exposure has also been associated with slight changes in blood components thought to be involved in the pathogenesis of atherosclerosis, such as endothelial cell count, platelet aggregate ratio, and platelet sensitivity to prostacyclin [Exs. 4–40, 4–80]. Many effects of ETS exposure, such as ischemia, may be additionally aggravated by simultaneous exposure to other compounds, such as solvents [Exs. 3–446, 4–99].

(e) Chronic Heart Effects. The occurrence of coronary heart disease in ETS-exposed nonsmokers has been studied by various epidemiological researchers [Exs. 4–85, 4–120, 4–122, 4–138, 4–139, 4–142, 4–148, 4–154, 4–191, 4–277, 4–295]. Small, but statistically significant (at $p \leq 0.05$), increases in coronary heart disease mortality [Exs. 4–85, 4–138, 4–139, 4–142, 4–277] indicate a modest impact of long-term ETS tobacco smoke exposure on the cardiovascular health of nonsmokers. The relative risks calculated in these studies ranged from 1.3 to 2.7.

The ability of ETS exposure to induce coronary heart disease has also been studied in animals. Zhu et al. [Ex. 4–330] exposed rats to ETS and showed a dose-related increase in myocardial infarct size and a decrease in bleeding time. But there were no significant differences in serum triglycerides, high density lipoprotein and cholesterol. This study showed that air nicotine, carbon monoxide, and total particulate concentrations increased with ETS exposure, and this increased exposure led to a continuous increase in plasma carboxyhemoglobin, nicotine, and cotinine levels in ETS-exposed rats. There was a positive relationship between the infarct size and air nicotine, carbon monoxide, total particulate concentrations and plasma carboxyhemoglobin, nicotine, and cotinine levels. The average concentrations of air nicotine, carbon monoxide and particulates, according to the authors, were 30-fold, 3-fold and 10-fold higher, respectively, than in a heavy smoking environment. The duration of exposure, however, was short compared to even a rat's lifetime. Infarct size nearly doubled following only 180 hours of ETS exposure distributed over a six week period.

In the same study, the effect of ETS exposure on platelet function and aortic and pulmonary artery atherosclerosis in New Zealand male rabbits was demonstrated. The increase of atherosclerosis after exposure to ETS was shown to be independent of changes in serum lipids and exhibited a dose-response relationship in this study. Average air nicotine, carbon monoxide and total particulate concentrations were 1,040 $\mu g/m^3$, 60.2 ppm and 32.8 mg/m^3 for high dose group and 30 $\mu g/m^3$, 18.8 ppm and 4.0 mg/m^3 for low dose group and <1 $\mu g/m^3$, 3.1 ppm and 0.13 mg/m^3 for the control group. Atherosclerosis in this study was significantly increased in the high dose group.

Olsen [Ex. 245] exposed rats daily to smoke from University of Kentucky 2R1 Reference cigarettes for 10 minutes, 7 times a week for 4, 8 or 20 weeks. Sidestream (SS) smoke was collected by a moving column of air spiked every minute with a puff of fresh mainstream (MS) smoke. Rats were exposed to this SS smoke collected in a 2 L/min air flow using a glass container placed over a burning cigarette. A fraction of this air flow containing SS smoke was diluted with fresh room air and continuously diverted to the rats as follows: 50%, 25% and 10% SS smoke. Carboxyhemoglobin content for each treatment group was determined immediately after the last smoke exposure and percent carboxyhemoglobin for each group was found to be: 4 week exposure-mainstream=7.2±1.2 and 25% sidestream=11.8±0.7; 8 week exposure mainstream=6.1±1.2 and 25% sidestream=11.9±0.9; 20 week exposure mainstream=8.3±0.9, 10% sidestream=6.30±0.5, 25% sidestream=10.8±0.8 and 50% sidestream=18.3±1.2. This indicates a tobacco smoke-related detrimental effect on blood components, thus increasing the probability that coronary disease would develop over a longer exposure period.

Research has shown that passive exposure to tobacco smoke damages endothelial cells and increases the number of circulating anuclear carcasses of endothelial cells [Ex. 4–80]. ETS appears to alter cardiac cellular metabolism in such a way that renders the myocyte less capable of producing adenosine triphosphate (ATP). Reduced oxidative phosphorylation in cardiac mitochondrial fractions taken from rabbits exposed to ETS has been demonstrated [Ex. 4–130]. Studies have indicated that the reduction in mitochondrial respiration secondary to ETS exposure is likely due to decreased cytochrome oxidase activity [Exs. 4–130, 4–131].

Nicotine, a component of tobacco smoke, has been shown in *in vitro* studies, to inhibit the release of prostacyclin, through inhibition of cyclooxygenase, from the rings of rabbit or rat aorta. Nicotine could also affect platelets by releasing catecholamines which lead to increased thromboxane A2 [Ex. 4–25]. Passive smoke also increases blood viscosity and hematocrit due to relative hypoxia induced by chronic carbon monoxide exposure [Ex. 4–25]. Nicotine, contained in cigarette smoke can lead to catecholamine release, which enhances platelet adhesiveness and decreases the ventricular fibrillation threshold. This threshold is also affected by carbon monoxide levels [Exs. 4–25, 4–196]. Cigarette smoke also increases the lipolysis that increases levels of plasma free fatty acids, which result in enhanced synthesis of LDL [Ex. 4–234].

In conclusion, there are multiple pathways by which ETS may damage the heart. ETS exposure has been demonstrated to both increase myocardial oxygen demand and decrease myocardial oxygen supply. If oxygen demand exceeds supply for a long enough period of time, then myocardial cell injury or even cell death can occur. In addition, ETS exposure may cause platelets to become less sensitive to the anti-clotting regulatory substances in the blood and therefore increase the tendency of the blood to clot. An increased tendency for the blood to clot may lead to an increased susceptibility to heart attacks.

ETS exposure may also contribute to the chronic formation of arterial wall plaques which are implicated in the event of an acute myocardial infarction. The two mechanisms described by which ETS exposure may stimulate plaque formation are endothelial cell injury and increased platelet activation.

Different people will have different abilities to deal with the increased stress on the heart and the increased tendency of the blood to clot as a result of ETS exposure. For example, a young, otherwise healthy individual may be able to tolerate short-term ETS exposure without apparent difficulty, although

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concluding that ". . . the OSH Act is designed to be protective of workers and is to protect against impairment with less impact than severe impairment."[5] The less severe conditions, such as upper respiratory symptoms and severe headaches, caused by poor indoor air quality are the same type as the PELs preamble concluded were material impairments. These specific conclusions of the Agency with respect to what constitutes material impairments were upheld by the Court of Appeals on review[6] although the Court disagreed with OSHA on other matters.

Therefore OSHA concludes that the adverse health effects caused by poor indoor air quality, which range from legionellosis to severe headaches to upper respiratory symptoms are material impairments of health which the Act allows the Agency to regulate.

The effects of the pneumonia caused by *Legionella* are deadly or severe. Although the rate of risk may not be as large as 1/1000 because the number of employees at risk is large. This effect alone makes a substantial contribution to a finding of significant risk, especially when taking into account the large number of cases.

As to the severe headaches, the Agency estimates that the excess risk of developing the type of non-migraine headache which may need medical attention or restrict activity which has been associated with poor indoor air quality is 57 per 1,000 exposed employees. In addition the excess risk of developing upper respiratory symptoms which are severe enough to require medical attention or restrict activity is estimated to be 85 per 1,000 exposed employees. These numbers are extrapolated from actual field studies and therefore show the magnitude of the problem at present. There is no doubt that better maintenance of ventilation systems such as required in the proposal will improve the quality of air in covered workplaces and reduce the number of cases. In addition the types of good practices prescribed in the proposal will substantially reduce the type of microbial contamination associated with Legionnaire's disease. Therefore, OSHA concludes that this number of less severe effects along with the severe effects from Legionnaire's disease, together, constitute a significant risk. Accordingly, OSHA preliminarily concludes that, the proposal will substantially reduce a significant risk of material impairment of health from poor indoor air quality.

[5] See discussion, 54 FR at 2361–2362

[6] See *AFL–CIO* v. *OSHA*, 965 F. 2d 962, 975 (11th Cir., 1992). The Court noted that "section 6(b)(5) of the Act charges OSHA with addressing all forms of 'material impairment of health *or functional capacity*,' and not exclusively 'death or serious physical harm' . . . from exposure to toxic substances."

VI. Preliminary Regulatory Impact Analysis

A. Introduction

Executive Order 12886 requires a Regulatory Impact Analysis and Regulatory Flexibility Analysis to be prepared for any regulation that meets the criteria for a "significant regulatory action." One of these criteria, relevant to this rulemaking is that the rule have an effect on the economy of $100 million or more per year. Based upon the preliminary analysis presented below, OSHA finds that the proposed standard will constitute a significant regulatory action.

The estimates presented in this Phase 1 Preliminary Regulatory Impact Analysis demonstrate technological and economic feasibility of the proposed standard. The analysis provides a non-detailed preliminary count of the affected employees and buildings, the associated costs, and benefits of the proposed standard provisions.

OSHA estimates the annual cost of compliance with the IAQ standard to be $8.1 billion, of which the most costly provision will be for the building systems operation and maintenance, $8.0 billion. The cost for eliminating exposure to ETS may range from $0 to $68 million depending on whether establishments ban smoking or allow smoking in designated areas. In order to assess the overall economic impact of the rule, OSHA also estimated the cost savings to employers, or cost savings that will result from the implementation of the proposed standard. The major forms of these savings are efficiency and productivity improvements, cost reductions in operations and maintenance, and reduced incidence of property damage. Cost savings associated with productivity improvements are estimated to be $15 billion annually.

OSHA preliminarily estimates that the proposed standard will prevent 3.0 million severe headaches and 4.5 million upper respiratory symptoms over the next 45 years. This is, approximately, 69,000 severe headaches and 105,000 upper respiratory symptoms per year. These estimates understate the prevalence of building-related symptoms since they reflect excess risk in only air conditioned buildings. In addition, 5,583 to 32,502 lung cancer deaths and 97,700 to 577,818 coronary heart disease deaths related to occupational exposure to ETS will be prevented over the next 45 years. This represents 140 to 722 lung cancer deaths per year and 2,094 to 13,001 heart disease deaths per year.

B. Industry Profile

The environmental concern for air pollution has been largely focussed on questions of outdoor air contamination. Recently, however, attention has begun to shift to concerns about the quality of air within buildings since people spend 80 to 90 percent of their time indoors [Ex. 3–1075H].

Indoor air is a variable complex mixture of chemicals and airborne particles. Its composition largely depends on the outdoor environment (urban or rural area), the shelter itself (age, construction material, electric equipment, heating, cooling, and ventilation systems), the activities of the occupants (smoking, nonsmoking, cooking by gas, oil or electricity) and the presence of plants and animals.

The Industry Profile chapter characterizes the building stock and describes the factors that affect indoor air quality. This section also presents the number of employees who work in buildings whose indoor air will be affected by the proposed standard.

1. Affected Industries

The standard covers all OSHA regulated industries: Agriculture, Oil and Gas Extraction (SIC 13), Manufacturing, Transportation, Communications, Wholesale Trade, Retail Trade, Finance, Insurance and Real Estate and Services. The scope of the proposal is twofold. The proposed indoor air quality compliance provisions would only cover employers with non-industrial work environments This includes public and private buildings, schools, healthcare facilities, offices and office areas. Coverage also applies to nonindustrial work environments that are part of industrial worksites (e.g., an office, cafeteria, or break room located at a manufacturing facility).

The provisions for protecting the nonsmoking employees from exposure to ETS apply to all indoor or enclosed work environments, in industrial and nonindustrial establishments. This would include maritime, construction, and agricultural workplaces.

2. Indoor Contaminants-Sources

Indoor air contaminants emanate from a broad array of sources that can originate both outside of structures as well as from within a building. When a building is new, some contaminants are given off quickly and soon disappear.

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disease. Studies that were determined to be "positive" by OSHA's review standards met standard epidemiologic and statistical criteria to support causation.

Overall, on the basis of the studies reviewed, OSHA concludes that the relative risk of lung cancer in nonsmokers due to chronic exposure to ETS ranges between 1.20 and 1.50 and the relative risk for heart disease due to ETS exposure ranges between 1.24 and 3.00.

TABLE IV–1.—EPIDEMIOLOGIC STUDIES REVIEWED BY OSHA—LUNG CANCER

Positive	Equivocal positive trend	Equivocal
Brownson et al. (1992)	Akiba et al	Brownson et al. (1987).
Correa et al	Butler	Buffler et al.
Fontham et al	Gao et al	Chan and Fung.
Garfinkel et al	Gillis et al	Hole et al.
Geng et al	Kabat and Wynder	Janerich et al.
Hirayama 1984a		Katada et al.
Humble		Koo et al.
Inoue et al		Lee et al.
Kalandidi et al		Shimizu et al.
Lam et al		Sobue et al.
Pershagen et al		Svenson et al.
Sandler et al		Wu et al.
Stockwell et al		
Trichopoulos et al		

TABLE IV–2.—EPIDEMIOLOGIC STUDIES REVIEWED BY OSHA HEART DISEASE

Positive	Equivocal positive trend	Equivocal
Dobson et al	Gillis et al	Garland et al.
He 1989	Hole et al	Lee et al.
Helsing et al	Humble et al	
Sandler et al	Svendsen et al	
Hirayama 1964		

Other relative risk estimates based on summaries of studies on ETS exposure performed by independent scientists and other government agencies are found in Tables IV–3 and IV–4. OSHA is not aware of any published risk assessments for overall exposure to indoor air pollutants.

TABLE IV–3.—PUBLISHED RISK ESTIMATES FOR LUNG CANCER

Study	Estimates of relative risk[1]
Daleger et al. [Ex. 4–78]	1.47 (.076–2.83)
NRC 1986 [Ex. 4–239]	1.34 (1.18–1.53)
Repace and Lowry [Ex. 4–263]	2.4
Vainio and Partanen [Ex. 4–312]	1.25–1.30
Wald et al. [Ex. 4–315]:	
Case-control studies	1.27 (1.05–1.53)
Prospective studies	1.44 (1.20–1.72)
Combined	1.55 (1.19–1.54)
Wells [Ex. 4–319]	2.10 (1.30–3.20)
EPA 1992 [Ex. 4–311]	[2] 1.19

[1] Numbers in parenthesis indicate published 95 percent confidence intervals.
[2] Pooled studies.

TABLE IV–4.—PUBLISHED RISK ESTIMATES FOR HEART DISEASE

Study	Estimates of relative risk
Steenland [Ex. 4–292]	[1] 1.51
	[2] 1.37
Wells [Ex. 4–319]	[3] 1.32

[1] Represents risk to nonsmoking men with spousal exposure.
[2] Represents risk to nonsmoking women with spousal exposure.
[3] Women.

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Most published risk assessments are based on spousal exposure to ETS. These studies have examined the lung cancer risk in nonsmoking housewives, using spousal smoking as a surrogate for the wife's exposure to ETS. The size of the association between these health effects and ETS exposure in the workplace is expected to be at least as large as the association seen between these health effects and ETS exposure in residential settings or public places. As

Source: https://www.industrydocuments.ucsf.edu/docs/pxhl0000

noted by Meridian Research in their 1988 report, ". . . it is the exposure to environmental tobacco smoke, and not the environment in which that exposure occurs, that is the important risk factor" [Ex. 4–221]. Therefore, health effects observed and the risk estimates calculated from studies of the general population, or of selected subgroups, such as nonsmoking wives of smoking husbands, are relevant to the working nonsmoking population.

In developing risk estimates for disease attributable to occupational exposure, reliance is placed on exposure encountered in the workplace to the extent possible. However, in the absence of purely occupational data, information derived in environments other than work sites is also considered. OSHA believes that there is no physiological difference related to exposure (or its outcome) regardless of where it is experienced. This is true regardless of whether the endpoint is lung cancer, heart disease, or indoor air related acute irritant effects. The only difference is that the degree of exposure may be greater in one place than in the other. Available information which uses nicotine concentration as an index of exposure suggests that the differences in exposure between office workplaces and residences lie well within the uncertainties of the determinations and for some workplaces, such as restaurants and transportation facilities, exposures are significantly higher than the average exposures found in residences. Thus, risk estimates based on residential exposures are expected to accurately reflect occupational risks in most workplaces and possibly underestimate the risk in some workplaces.

In developing its risk assessment for lung cancer, the EPA reviewed 19 studies which investigated nicotine concentrations in various environments [Ex. 4–311]. EPA's analysis showed that the range of average nicotine concentrations in office workplaces is very similar to that of homes. However, in some workplaces, such as restaurants and transportation facilities, exposures are significantly higher. It is true that there are many complicating factors in such determinations which could affect any final conclusions. For example, it is important to consider the duration of exposure, the intensity of exposure, the distance from the sources and other factors as well. However, EPA's analysis suggests that risk assessments based on home exposures are relevant to workplaces as well and, in comparison to some workplaces, may even result in an underestimate of the true occupational risk.

In addition, other studies substantiate the magnitude of workplace exposures. For example, Emmons et al. [Ex. 4–98] found that the majority of ETS exposure occurred in the workplace. Study subjects were selected from workplace settings with a wide range of ETS exposure. The work sites ranged from those with minimal restriction of smoking and high levels of exposure to work sites with extensive smoking restrictions and low exposure. Ninety percent of the subjects worked outside the home. Eighty-four percent of those who worked outside the home (75.6% of the total sample) reported being regularly exposed to smoking in the workplace. While the most highly exposed individuals in the study were those who had both home and work exposures, it is clear that workplace exposure constituted a significant component of overall exposure. Subjects who did not live with smokers reported that the majority of their exposure was in the workplace (mean=36.1 min/day), home (mean=1.4 min/day) or in other locations (mean=13.1 min/day). Subjects who lived with smokers reported receiving slightly more exposure at home than the workplace, however the difference between home exposure and workplace exposure was not substantial (work: mean=29.4 min/day, home: mean=31.2 min/day, other: mean=27.1 min/day). These results are shown in Table IV–5. The importance of the findings from this study is twofold. First, it indicates that the workplace is the primary source of ETS exposure for nonsmokers, who do not live with smokers. Secondly, it shows that for nonsmokers living with smokers, even though their household environment becomes their primary source of exposure, the workplace still contributes a substantial amount of exposure, comparable to that experienced by the nonsmoker living with nonsmokers (29.4 min/day v. 36.1 min/day).

TABLE IV–5.—EXPOSURE TO ETS BY LOCATION [1]

Subject Category	Exposure (min/day)	95 percent confidence interval
Living with a smoker:		
Workplace	29.4	(7.01–51.80)
Home	31.2	(21.60–40.80)
Other	27.1	(15.10–39.10)
Living without a smoker:		
Workplace	36.1	(22.70–49.50)
Home	1.4	(0.05–2.75)
Other	13.1	(8.75–17.40)

[1] Source: Emmons et al. [Ex. 4–98]

Cummings et al. [Exs. 4–67], Hudgafvel-Pursiainen et al. [Ex. 4–152], and Marcus et al. [Ex. 4–205] also present results to show significant workplace exposures to ETS. A re-analysis of the CAPS data (a detailed description of this study is found in the EXPOSURE section) shows that the workplace contributes on the average 46 percent to the total ETS exposure experienced by a nonsmoking worker.

C. Data Sources

As mentioned previously, only diseases that have been reported to be significantly associated with ETS exposure and for which OSHA has access to data will be used in calculating health risk due to occupational exposure to ETS. These will be referred to as the "diseases of interest" and include coronary heart disease (excluding strokes) as defined in the Framingham study and lung cancer.

Ideally, data on the incidence of the diseases of interest in the U.S. population were needed to estimate the number of cases of disease in employed nonsmokers. Since nationwide incidence data were not available for nonsmokers, several survey sources were used to estimate the mortality rates for heart disease (Framingham Community Study) [Ex. 4–108], and lung cancer (Cancer Prevention Survey conducted by the American Cancer

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Source: https://www.industrydocuments.ucsf.edu/docs/pxhl0000

drip-pans in HVAC systems, drainage pans in refrigerators, dirty heating coils, and are also associated with mammals, arthopods and insects. Table III–3 gives examples of biologicals found in indoor environments.

Various allergens have been associated with the development of allergic rhinitis, asthma, or airway hyperresponsiveness (Table III–3) [Ex. 4–33]. Many of these allergens are common to the nonindustrial work environment. These include chemical volatiles and dusts, arthropods, and dusts, particulates & fibers.

TABLE III–3.—EXAMPLES OF BIOLOGICALS FOUND IN INDOOR ENVIRONMENTS [1]

Class	Agent or component	Origin
Arthopods and Insects	Whole organism, body parts, feces	Furnishings, building materials, food.
Microbes:		
Algae	Whole organism, cellular components	Outdoor air, HVAC (rare).
Bacteria	Whole organism, spores and cell walls, endotoxin.	Stagnant water, floods, cooling towers, industrial processes.
Fungi	Whole organism spores and hyphae toxins and volatiles.	Moist surfaces, HVAC system, bird droppings, outdoor air.
Protozoa	Whole organism cellular components	Water reservoirs, pets (rare).
Viruses	Whole organism	Humans and pets (rare).
Pets	Skin, scales danders, urine, saliva, feces	Pets, pet litter, pet cages, pet toys, pet bedding.
Plants	Stems, leaves and pollens	Outdoor and indoor air.

[1] Adapted from Ex. 4–33.

TABLE III–4.—INDOOR AIR ALLERGENS ASSOCIATED WITH ASTHMA [1]

Class	Typical examples
Animal:	
Avian	High and low molecular weight proteins from feathers and droppings.
Canine and Feline	High and low molecular weight proteins from dander, saliva, and feces.
Arthropods:	
Mites, Cockroaches, Crickets and Moths	Structural proteins, carbohydrates and metabolites.
Dusts, Particulates and Fibers:	
Household	Pollens, fungi, danders and mites.
Metal	Chromium, cobalt, nickel, platinum, and vanadium.
Plant	Castor bean, coffee, cotton, flour, and grain.
Wood	Oak, mahogany, redwood, red cedar.
Chemical Volatiles and Dusts	Acrylates, amines, anhydrides, colophony, enzymes, epoxy resins, freon, furfuryl alcohol, resins, isocyanates, latex, organophosphates, polyvinyl chloride, vegetable gums.
Microbes and Microbial Products:	
Bacteria	*Bacillus* spp.
Fungi	*Alternaria* spp., *Aspergillus* spp., *Botrytis* spp., *Cladosporium* spp., *Penicillium* spp., *Pullularia* spp.
Pollens	*Agrostis* spp., *Alopecurus* spp., *Anthoxanthum* spp. *Cynosurus* spp., *Dactylis* spp., *Holcus* spp., *Lolium* spp., *Secale* spp.

[1] Source: Ex. 4–33.

Exposures that cause hypersensitivity reactions include microorganisms, fumes, vapors, and dusts (Table III–5). These exposures are associated with the development of hypersensitivity pneumonitis or a less serious variant, humidifier fever [Ex. 4–33]. Many of these contaminants are found in the nonindustrial workplace. Birds and rodents are common pests. Air intakes can be contaminated with bird droppings and other avian-associated problems when used as nesting sites. These problems can affect the quality of the air being brought into the ventilation system through these air intakes. Rodent infestations affect work areas directly. Many of the chemicals listed in Table III–5 are commonly found in most workplaces.

In summary, exposure to contaminants in nonindustrial workplaces will vary according to the characteristics of the building. These include its age, types of materials used in construction and the type of equipment and supplies that are used by building occupants. The design, maintenance, and operation of the building's HVAC system as well as the general housekeeping of the building, can greatly influence the levels of contaminants that exist.

OSHA requests data on the levels of these contaminants in nonindustrial workplaces.

TABLE III–5.—INDOOR AIR CONTAMINANTS ASSOCIATED WITH HYPERSENSITIVITY PNEUMONITIS [1]

Class	Typical examples
Animals:	
Avian	High and low molecular weight proteins from feathers and droppings.
Rodent	Low molecular weight proteins from urine and feces.
Arthropods:	
Weevils	*Sitophilus* spp.

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Source: https://www.industrydocuments.ucsf.edu/docs/pxhl0000

steam bugs near the coffee shop and on back stairs. The comment continued that "a sink faucet in the lunch room has been leaking for years and water runs on the counter under the toaster and microwave. The water heater had leaked for about 2 months before it was fixed. At that time the carpet was soaked and water was running under the wall into a supervisor's office. There is a moldy odor from this carpet and the floor below."

Cancer has also been reported to be associated with poor indoor air quality. A courthouse in San Diego, California [Ex. 3–55], "is notorious for poor air quality and employee respiratory illness and cancer." It was reported to OSHA that many long-term employees have cancer (stomach and lung cancer), terminal lung disease, chronic ear and throat infections, and bronchial problems" [Exs. 3–585, 3–635, 3–637, 3–68].

Comment 3–630 from a union reported that "[a]fter surveying thousands of workers across the country, SEIU compiled actual survey responses that list adverse health effects caused by indoor air pollution. These include headaches, nose congestion or irritation, throat irritation, dry cough, dry or itchy skin, dizziness, nausea, lethargy or fatigue, colds, asthma/wheezing, chest tightness, runny nose/post nasal drip, eye or contact lens irritation, respiratory difficulties. In addition, EPA estimates that pollutants found in indoor air are responsible for 2,500 to 6,500 cancer deaths each year" [refer to Ex. 3–630L].

These concerns are not just relevant to office workers but also to maintenance and other nonindustrial workers that work in indoor environments. For example, comment 3–347 responded that "[i]n our closed, indoor work environments, air quality is a very real health and safety concern to professional painters. I have seen firsthand otherwise healthy men and women pass out or get violently ill as a result of being exposed to indoor air contaminants." Comment 3–412 responded "[o]ur locals have encountered air-pollution problems ranging from ink mist and photocopier emissions to asbestos and microbial disease. The level of toxic chemical contaminants is often alarmingly high in our darkrooms, and carbon-monoxide emissions from trucks at newspaper loading docks frequently penetrate the ventilation system. In 1985 microbial contamination from a water tower infected six New York Times employees with Legionnaires' Disease and 34 others with less serious respiratory infections."

Operation engineers are also affected by poor indoor air quality. Comment 3–452 responded that "[t]his is particularly important for the operation engineers who appear healthy and then suffer from respiratory problems, much like allergic reactions, after working in a building with poor ventilation."

2. Environmental Tobacco Smoke

Many case reports of severe material impairment of health due to occupational exposure to ETS have been reported to OSHA through submission to the indoor air quality docket [H–122]. Information contained in these comments indicate that adverse health effects in workers due to environmental tobacco smoke exposure while at work range from mucous membrane irritation (eye, nose, and throat effects) to more severe, life-threatening conditions, such as status asthma, other chronic lung diseases and heart diseases. For example, comment 3–309 responded [Regarding ETS exposure in a cafeteria], "By the time I have finished lunch my eyes are tearing, my nose is plugged, and I have a headache" as well as comment 3–315, "I had fewer headaches and fewer respiratory ailments; my chronic sore throat disappeared [after a company-wide no smoking policy was implemented]". Comment 3–22 responded "[m]y patients find it hard to obtain smoke free workplaces. I have seen patients who have suffered status asthma from workplace smoking, patients who have had to quit their jobs because of ETS in the workplace. Recently, one of my never smoking patients sustained vocal cord lesions seen almost entirely in smokers." Comment 3–104 continued that "[p]assive tobacco smoke (PTS) is the principal indoor air contaminant in my office building in Rockefeller Center. While smoking is limited to 'private offices', the smoke flows freely from these private offices throughout the entire general office areas since the smokers will not keep their doors closed, and even when they do, they have to come out sometime. And, as soon as the door is opened, the dense smoke accumulation within the office is diffused to all adjacent work areas. Because office buildings have closed ventilation systems, only a 'smoke free' office policy can be effective. Half measures only cause further stress, frustration and irritation to both smokers and nonsmokers." Comment 3–289 responded that "I have been exposed to asbestos culminating in my getting asbestosis (plural plaque) of the lungs. The combination of asbestos exposure plus second-hand smoke from my smoking co-workers has posed and is currently posing a health risk to me."

III. Exposure

Contaminants which contribute to poor indoor air quality can be attributed to both outside air and inside air. Outside air contaminants can be introduced into a building through the ventilation intakes, doors, building envelope, and windows. Outside air contaminants include vehicular exhausts, industrial emissions, microbiologicals, and pollen. Inside air contaminants are emitted from building materials and furnishings, appliances, office equipment and supplies, biological organisms, and of course, pollutants introduced by the building occupants themselves. Inside air contaminants include tobacco smoke, volatile organic compounds, combustion gases such as carbon monoxide, and occupant-generated bioeffluents. The concentration of these contaminants in buildings can increase if ventilation systems are inadequately designed, maintained and operated or if strong local contaminant sources are not controlled.

A. Sources of Indoor Air Contaminants

A wide variety of substances are emitted by building construction materials and interior furnishings, appliances, office equipment, and supplies, human activities, and biological agents. For example, formaldehyde is emitted from various wood products, including particle board, plywood, pressed-wood, paneling, some carpeting and backing, some furniture and dyed materials, urea-formaldehyde insulating foam, some cleaners and deodorizers, and from press textiles. Volatile organic compounds, including alkanes, aromatic hydrocarbons, esters, alcohols, aldehydes, and ketones are emitted from solvents and cleaning compounds, paints, glues, caulks, and resins, spray propellants, fabric softeners and deodorizers, unvented combustion sources, dry-cleaning fluids, arts and crafts, some fabrics and furnishings, stored gasoline, cooking, building and roofing materials, waxes and polishing compounds, pens and markers, binders and plasticizers. Pesticides also contain a variety of toxic organic compounds.

Building materials are point sources of emissions that include a variety of VOCs (Table III–1). Some of these materials have been linked to indoor air quality problems. The probability of a source emitting contaminants is related to the age of the material. The newer the material, the higher the potential for emitting contaminants. These materials

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TABLE III–7.—PARTICULATE PHASE CONSTITUENTS OF TOBACCO SMOKE AND RELATED HEALTH EFFECTS—Continued

Constituent	Amount in MS	Ratio in SS/MS	Health effects
NNK [4-(N-methyl-N-nitrosamino)-1-(3-pyridyl)-1-butanone]	100–1000 ng	1–4	N/A.[5]
N-nitrosodiethanolamine	20–70 ng	1.2	Probable human carcinogen.[4]
Cadmium	110 ng	7.2	Probable human carcinogen.[4]
Nickel	20–80 ng	13–30	Known human carcinogen.[4]
Zinc	60 ng	6.7	Irritant, nausea, vomiting.[2]
Polonium-210	0.04–0.1 pCi	1.04.0	Known human carcinogen.[4]
Benzoic acid	14–28 µg	0.67–0.95	Irritant.
Lactic acid	63–174 µg	0.5–0.7	Irritant.[3]
Glycolic acid	37–126 µg	0.60.95	Irritant.[2]
Succinic acid	110–140 µg	0.43–0.62	N/A.[5]
PCDD's and PCDF's[6]	1 pg	2	N/A.[5]

[1] NIOSH Pocket Guide to Chemical Hazards. U.S. Department of Health and Human Services. Public Health Services, 1990. Ex. 4–238.
[2] The Merck Index, 10th Edition, Merck & Co., Inc., 1983. Ex. 4–220.
[3] Hazards in the Chemical Laboratory. Ed: L. Bretherick, The Royal Society of Chemistry, 1986. [Ex. 4–137]
[4] EPA: Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders, 1992. [Ex. 4–311]
[5] N/A—Relevant information not available.
[6] PCDDs—Polychlorinated dibenzo-p-dioxins; PCDFs—Polychlorinated dibenzofurans.

MS and SS cigarette smoke are chemically and physically complex mixtures consisting of electrically charged submicron liquid particles at very high concentration consisting of permanent gases, reactive gases, and a large variety of organic chemicals. The composition of the smoke and especially the total quantities of individual constituents delivered are dependent on the conditions of smoke generation [Ex. 4–311].

Nicotine, while found in the particulate phase in MS, is found predominantly in the gas phase in ETS [Ex. 4–100]. The differences in size distribution for MS and SS particles, as well as the different breathing patterns of smokers and nonsmokers, affect deposition of the produced particle contaminants in various regions of the respiratory tract.

There are substantial similarities and some differences between MS and SS emissions from cigarettes [Exs. 3–689D, 4–129, 4–239]. Differences in MS and SS emissions are due to differences in the temperature of the combustion of tobacco, pH, and degree of dilution with the air, which is accompanied by a correspondingly rapid decrease in temperature. SS is generated at a lower temperature (approximately 600°C between puffs versus 800 to 900°C for MS during puffs) and at a higher pH (6.7–7.5 versus 6.0–6.7) than MS. Being slightly more alkaline, SS contains more ammonia, is depleted of acids, contains greater quantities of organic bases, and contains less hydrogen cyanide than MS. Differences in MS and SS are also ascribable to differences in the oxygen concentration (16% in MS versus 2% in SS). SS contaminants are generated in a more reducing environment than those in MS, which will affect the distribution of some compounds. Nitrosamines, for example, are present in greater concentrations in SS than in MS.

Many of the compounds found in MS, which were identified as human carcinogens, are also found in SS emissions [Exs. 3–689D, 4–93, 4–129, 4–239, 4–269] and at emission rates considerably higher than for MS. SS contains ten times more polycyclic aromatic hydrocarbons, aza-arenes and amines as compared with MS [Ex. 4–126]. All of the five known carcinogens, nine probable human carcinogens, and three animal carcinogens are emitted at higher levels in SS than in MS, several by an order of magnitude or more. Several toxic compounds found in MS are also found in SS (carbon monoxide, ammonia, nitrogen oxides, nicotine, acrolein, acetone, etc.), in some cases by an order of magnitude or higher (Tables III–6 and III–7).

SS emissions, quantitatively, show little variability as a function of a number of variables (puff volume, filter versus nonfilter cigarette, and filter ventilation [Exs. 4–1, 4–34, 4–54, 4–128, 4–129, 4–141]. The lack of substantial variability in SS emissions is related to the fact that they are primarily related to the weight of tobacco and paper consumed during the smoldering period, with little influence exerted by cigarette design [Ex. 4–129].

(b) Human Activity Pattern Studies Used to Assess Workplace Exposure. Human activity pattern studies utilize random samples of human activity patterns using questionnaires and time-diary data to provide detailed generalizable data about human behavior. Such studies have been used to assess exposure to ETS. In 1987–1988, the California Air Resources Board sponsored a probability-based cross-sectional sample of 1,579 Californians aged 18 years and older, called the California Activity Pattern Survey (CAPS) [Exs. 4–168, 4–271]. The study was designed to provide information on time spent in various locations, including indoors, outdoors, and in transit, as well as specific microenvironments, such as living rooms, kitchens, automobiles, or buses. The study focused on time spent in activities such as cooking or playing sports, but more specifically targeted activities and environments that had implications for air pollution exposure, such as the presence of smokers, use of cooking equipment or solvents.

In analyzing the data from CAPS, Jenkins et al. [Ex. 4–168] and Robinson et al. [Ex. 4–271] found that time spent at work had a high correlation with exposure to ETS. This association of ETS exposure with work settings remained strong after controlling for the length of the activity episode, and hence was not simply a function of longer time intervals at work. Robinson et al. [Ex. 4–271] also found that men reported higher levels of exposure than women, even after controlling for age, employment status, shorter working hours, etc. This finding suggests that the epidemiological studies of passive smoking and lung cancer, which have focussed on women, may be underestimating the effect of ETS on lung cancer.

Further analysis of the CAP study [Ex. 4–169] verifies the high percentage of nonsmokers who are exposed to ETS while at work. This is indicated when the data are analyzed by employed nonsmoker status. As indicated in Table III–8, 51% of male and 38% of female nonsmokers reported ETS exposure at work. The average duration of this exposure was 313 minutes for males and 350 minutes for females. When the group that reported exposure at the

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OSHA believes that these impacts are not large enough to impair economic viability. While some marginal firms might be more seriously impacted, extensive economic dislocation is not expected to occur in any industry. OSHA has, therefore, preliminarily determined that the standard is economically feasible.

(2) Regulatory Flexibility Analysis

The proposed IAQ standard will affect numerous small establishments and a portion of these establishments may have difficulty financing the compliance actions needed to comply depending on which alternative they choose. This section examines the potential for exceptional impacts among small establishments.

The nature of compliance action limits the potential for exceptionally large compliance burdens on small businesses because most costs will be incurred on a per employee or per square foot basis. The number of buildings occupied with establishments with fewer than 20 employees is estimated at 3.7 million or 82 percent of all buildings. Of these, 76 percent have floor space less than 10,000 square feet. Thus, small firms will incur low costs because they have small floorspace and few employees.

To this point of the analysis, OSHA has not distributed the number of buildings across establishments since there are no data on which to describe the establishments in multi-tenant buildings. Therefore, OSHA developed establishment specific compliance costs based on the estimates presented in section E of this report. The economic impact by firm size is estimated with the assumption that all establishments will require HVAC maintenance. It was assumed that each establishment has a floor space of 10,000 square feet. To examine the potential regulatory burden that would be experienced by small establishments, OSHA calculated the ratio of their annual compliance cost to their sales and pre-tax profit for two scenarios for dealing with ETS: (1) provide designated smoking areas, or (2) totally ban smoking in the workplace. As shown in Table VI–14, for both scenarios, the average ratio of compliance costs to sales ranges from 0.44 percent to 0.52 percent. The highest impact (2.79 percent) for establishments not banning smoking would be in Personal Services (SIC–72). Estimates of compliance cost as a percentage of pre-tax profits were less than 7.05 percent for most sectors; Social Services establishments (SIC–83) would experience the largest reduction in profit (31 percent), if they allow smoking in designated rooms.

These estimates apply to the average firm in each sector. The degree to which affected firms will either incur or shift compliance costs depends largely on the competitive environment in which the establishments operate and on the elasticity of demand for the establishment's services and commodities. OSHA requests information regarding compliance costs against indicators of the demand for and the costs of the types of services and commodities provided by establishments which would be affected by the proposed standard. OSHA specifically requests comments, including empirical data regarding the demand elasticity of such establishments' patrons who will not be permitted to smoke in the presence of employees at such establishments. If economic feasibility is shown to be an issue for establishments such as bars and restaurants, what methods of compliance would adequately protect workers in a feasible manner?

(3) Environmental Impact

The provisions of the standard have been reviewed in accordance with the requirements of the National Environmental Policy Act (NEPA) of 1969 [42 U.S.C. 432, et seq.], the Council on Environmental Quality (CEQ) NEPA regulations [40 CFR Part 1500], and OSHA's DOL NEPA Procedures [29 CFR Part 11]. As a result of this review, OSHA concluded that this rule will have no significant environmental impact.

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TABLE III–5.—INDOOR AIR CONTAMINANTS ASSOCIATED WITH HYPERSENSITIVITY PNEUMONITIS [1]—Continued

Class	Typical examples
Mites	*Ascaris* spp.
Altered Host Proteins or Chemical Hapten-Carrier Conjugates.	Amines, anhydrides, epoxy resins vegetable gums, and isocyanates.
Microbes:	
Bacteria	*Thermoactinomycetes* spp., *Bacillus* spp.
Fungi	*Aspergillus* spp., *Auerobasillium* spp., *Cephalosporium* spp., *Penicillium* spp.
Organic Dusts & Particulates:	
Wood	Bark, Sawdust and Pollen.
Grain	Arthropod- and microbially-contaminated grains and flours.
Cleaning Products	Dust residues from carpet cleaning agents.

[1] Source: Ex. 4–33.

C. Exposure Studies

1. Low-level Contaminants

Experimental studies have demonstrated that exposure of susceptible people to low level mixtures of VOCs have induced mucous membrane irritation and pulmonary effects. Some of these studies are discussed below.

The potential of indoor air contamination to produce adverse effects in humans was demonstrated by Molhave et al. in Denmark [Ex. 4–20]. These researchers studied 62 subjects suffering from "indoor climate symptoms". These subjects reported primarily eye and upper respiratory tract irritation, but were otherwise healthy individuals that did not suffer from asthma, allergy, or bronchitis. The subjects were exposed to a mixture of VOCs in concentrations of 0, 5, or 25 mg/m^3. These concentrations respectively represented "clean" air, average polluted air, and the maximum polluted air in Danish households. After exposure, a Digit Span test was administered. The study found significant declines in performance on this test; demonstrating that low-level exposures to volatile organic compounds had an adverse effect on the ability to concentrate [Ex. 4–20].

Otto et al. [Ex. 4–248], repeating the Molhave et al. (1984) experiment, studied 66 healthy subjects with no history of eye and upper respiratory tract irritation. These subjects were exposed at 0 and 25 mg/m^3 VOC-contaminated air. Otto et al. reported that while subjects found the odor of chemicals unpleasant, to degrade indoor air quality, to increase headache, and produce general discomfort, VOC exposure for 2.75 hours duration did not affect performance on any behavioral tests. These results imply that persons who experience symptoms of SBS may have a lower threshold for certain health effects compared to nonreactive people. This suggests that those with compromised immune response (e.g. allergy sufferers) may be at elevated risk of SBS.

Ahlstrom, et al. [Ex. 4–2] found that synergistic effects may occur when one strong indoor irritant interacts with other indoor contaminants present at low-level concentrations. Ahlstrom et al. found that there was almost a 4-fold increase in the perceived odor strength of formaldehyde at low concentration (0.08 ppm) when mixed with 100% indoor air from a building where SBS was reported, relative to 10% indoor air from the same building.

The Report of the Canadian Interministerial Committee on Indoor Air Quality [Ex. 4–264] adopts the World Health Organization's definition of health: "Health refers to a state of complete physical, mental, and social well being, and not just the absence of disease or infirmity." This definition was adopted to allow the setting of indoor air quality guidelines based on "comfort" as well as "health". The report observes that the symptoms of SBS are sufficiently general or subjective that they may be indicative of several other medical conditions. Therefore, perhaps the best indicator that workplace exposure may play a role in the symptoms reported by an individual is the observation that symptoms worsen during the work day, and disappear shortly after leaving work. They state that because there is a wide variation in individual susceptibility, based on genetics, age, medication, previous exposure to pollutants, gender, and state of health, especially those with allergies, that certain individuals may be more sensitive to SBS than others.

2. Bioaerosols

The levels of bioaerosols in the indoor environment should reflect those found in the outdoor environment. A rank order assessment, comparing the abundance of microorganisms in the outdoor versus indoor environment is one way of assessing this relationship [Exs. 3–61, 4–229]. If indoor and outdoor sampling results are not comparable, then it is possible that a reservoir of a particular microbe may be amplifying in the indoor environment; especially if moisture and a nutrient-rich substrate are available [Ex. 4–229]. An example of this would be *Legionella*. Commonly found in the outdoor environment, the bacteria are as expected, commonly found in untreated potable and nonpotable water. Situations can occur that allow these reservoirs to amplify not only in potable water and hot water service systems but also water used in cooling towers and evaporative condensers [Ex. 4–229]. Infection occurs if the bacteria are disseminated, either through the HVAC system or potable water system (e.g., showers) to the breathing zone of a susceptible person. A healthy individual may develop the less severe Pontiac Fever. An individual that smokes or is older may develop the more serious pneumonia [Exs. 4–33, 4–229].

3. Environmental Tobacco Smoke

The burning of tobacco in enclosed workplaces releases an aerosol containing a large variety of solid, liquid, and gas phase chemical compounds. Generation of tobacco smoke is governed by the source emission characteristics of smokers and their tobacco products, whereas removal is primarily determined by the rate of replacement of building air by outside air, with re-emission of surface-sorbed compounds playing a minor role. Natural and mechanical ventilation systems are designed primarily to limit the accumulation of the products of human respiratory metabolism, and secondarily to limit odor; not to control the byproducts of biomass combustion. Thus, smoking indoors creates air pollution which is not adequately abated by customary ventilation systems.

Exposure to tobacco smoke primarily occurs through the inhalation route.

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A. Environmental Tobacco Smoke
B. Indoor Air Quality
VI. Preliminary Regulatory Impact Analysis
A. Introduction
B. Industry Profile
1. Affected Industries
2. Indoor Contaminants-Sources
3. Controlling Indoor Air
4. Building Characteristics
5. Profile of Affected Buildings
6. Buildings with Indoor Air Problems
7. Number of Employees Affected
8. Environmental Tobacco Smoke
(a) Smoking Ordinances and Policies
(b) Number of Nonsmokers Working Indoors
C. Nonregulatory Alternatives
1. Introduction
2. Market Imperfections
3. Alternative Nonregulatory Options
(a) Tort Liability
(b) Workers' Compensation
4. Conclusion
D. Benefits
1. Indoor Air Quality
2. Environmental Tobacco Smoke
3. Cost Savings
(a) Worker Productivity
(b) Property Damage, Maintenance and Cleaning Costs
E. Technological Feasibility and Compliance Costs
1. Technological Feasibility
2. Compliance Costs
(a) Developing Indoor Air Quality Compliance Programs
(b) Indoor Air Quality Operation and Maintenance Program
(c) Training for HVAC Maintenance Workers and Informing Employees About the Indoor Air Quality Standard
(d) Compliance with Related Standards
(e) Air Contaminant-Tobacco Smoke
(f) Air Quality During Renovation and Remodeling
F. Economic Impact and Regulatory Flexibility Analysis
1. Economic Feasibility
2. Regulatory Flexibility Analysis
3. Environmental Impact
VII. Summary and Explanation
A. Scope and Application: Paragraph (a)
B. Definitions: Paragraph (b)
C. Indoor Air Quality Compliance Program: Paragraph (c)
D. Compliance Program Implementation: Paragraph (d)
E. Controls for Specific Contaminant Sources: Paragraph (e)
F. Air Quality During Renovation and Remodeling: Paragraph (f)
G. Employee Information and Training: Paragraph (g)
H. Recordkeeping: Paragraph (h)
I. Dates: Paragraph (i)
J. Appendices: Paragraph (j)
K. Specific Issues
VIII. State-Plan Standards
IX. Federalism
X. Information Collection Requirements
XI. Public Participation
XII. List of Subjects in 29 CFR Parts 1910, 1915, 1926, and 1928
XIII. Authority and Signature
XIV. Part 1910, 1915, 1926, 1928—Proposed Occupational Safety and Health Standards

Supplementary Information

A. Events Leading to This Action

Concern about the health hazards posed by occupational exposure to environmental tobacco smoke (ETS) prompted three public interest groups to petition the Agency in May 1987 for an Emergency Temporary Standard under section 6(c) of the Occupational Safety and Health (OSH) Act, 29 U.S.C. 655(c). The American Public Health Association and Public Citizen submitted a joint petition; Action on Smoking and Health (ASH) also submitted a petition. The petitions requested the prohibition of smoking in most indoor workplaces.

OSHA determined, that available data with respect to exposures were insufficient to demonstrate the existence of a "grave danger," within the meaning of section 6(c) of the OSH Act, from workplace exposure to ETS. OSHA denied the petitions in September 1989 but continued to investigate regulatory options.

In October 1989 ASH filed suit in the U.S. Court of Appeals for the District of Columbia Circuit for review of OSHA's denial of its petition for an Emergency Temporary Standard. The court denied ASH's petition for review in May 1991, finding that OSHA has reasonably determined that it could not sufficiently quantify the workplace risk associated with tobacco smoke to justify an Emergency Temporary Standard.

OSHA issued on September 20, 1991, a Request for Information (RFI) (56 FR 47892) on indoor air quality problems, in order to obtain information necessary to determine whether it would be appropriate and feasible to pursue regulatory action concerning Indoor Air Quality (IAQ). Issues on which comments were requested in the RFI included health effects attributable to poor IAQ, ventilation systems performance, exposure assessment, and abatement methods. Information concerning specific contaminants such as ETS and bioaerosols was also requested.

In March 1992, the AFL–CIO petitioned OSHA to promulgate an overall IAQ standard. OSHA responded in May 1992 that such a standard was under consideration.

In response to the RFI, over 1,200 comments were submitted by interested persons, groups, unions, and industries. Issues of particular concern identified in the comments, in addition to health effects considerations, include the lack of ventilation performance standards; the lack of worker training on the operation and maintenance of Heating Ventilation and Air Conditioning (HVAC) systems; the lack of pollutant source control; and the lack of available technical guidance on IAQ issues and control techniques.

Of the comments that specifically addressed the question of whether OSHA should regulate IAQ, a majority (75%) indicate support for regulation. Of those that commented on the need for regulation, approximately 21% were explicitly in favor of a regulation on ETS, more than 41% were in favor of an overall IAQ regulation, and approximately 13% were in favor of a combined IAQ regulation.

Numerous comments focused on the adverse health effects of tobacco smoke and of general indoor air pollution. The health effects of concern relevant to both tobacco smoke and indoor air pollutants ranged from the acute irritant effects to cancer.

Comments submitted in response to the RFI indicated wide support for a regulatory approach that would focus on the design, operation and maintenance of building ventilation systems, source reduction methodology, and worker information and training programs. Commenters also recommended that provisions should require that employers receive training about the regulation and the need for compliance, and that their training regarding building HVAC maintenance and operation be tailored to the level of complexity of the HVAC system and their personal degree of involvement.

Many commenters particularly felt that regulation of IAQ was necessary to eliminate exposures to ETS in the workplace. Commenters urged the Agency to either ban smoking completely from the workplace or allow smoking only in separately ventilated, designated smoking areas that were separate from work areas.

OSHA believes that data submitted to the record, and other evidence, support the conclusion that air contaminants and other air quality factors can act to present a significant risk of material impairment to employees working in indoor environments. Adverse health effects associated with poor IAQ may include sensory irritation, respiratory allergies, asthma, nosocomial infections, humidifier fever, hypersensitivity pneumonitis, Legionnaires' disease, and the signs and symptoms characteristic of exposure to chemical or biologic substances such as carbon monoxide, formaldehyde, pesticides, endotoxins, or mycotoxins.

The Agency believes that available data support proposing regulation of IAQ, including exposure to ETS. Further stimulus for this determination was provided by conclusions reached in a

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Others continue off-gassing at a slow pace for years. Common office supplies and equipment have been found to release hazardous chemicals—especially duplicators and copiers. Bulk paper stores have been found to release formaldehyde [Ex. 3–1087A20]. Some typical contaminants are listed below:

(a) Gases and Vapors (organic/inorganic):
—Radon
—Sulfur dioxide
—Ammonia
—Carbon Monoxide
—Carbon Dioxide
—Nitrous Oxides
—Formaldehyde
(b) Fibers:
—Asbestos
—Fiberglass/Mineral Wools
—Textiles/Cotton
(c) Dusts:
—Allergens
—Household dust (mites)
—Pollens:
—Feathers
—Danders
—Spores
—Smoke/Fume
—Environmental Tobacco Smoke
—Coal
—Wood
(d) Microbes:
—Bacteria
—Fungi
—Viruses

People contribute millions of particles to the indoor air primarily through the shedding of skin scales. Many of these scales carry microbes, most of which are short lived and harmless. Clothing, furnishings, draperies, carpets, etc. contribute fibers and other fragments. Cleaning processes, sweeping, vacuuming, dusting normally remove the larger particles, but often increase the airborne concentrations of the smaller particles. Cooking, broiling, grilling, gas and oil burning, smoking, coal and wood generate vast numbers of airborne indoor pollutants in various classifications.

3. Controlling Indoor Air

Control of pollutants at the source is the most effective strategy for maintaining clean indoor air. However, control or mitigation of all sources is not always possible or practical. In the case of ETS, this means restricting smoking to separately ventilated spaces. General ventilation is, therefore, the second most effective approach to providing acceptable indoor air [Exs. 3–1061G, 3–1075J].

Outside air dilutes and removes contaminants through natural ventilation, mechanical ventilation or through infiltration and exfiltration. Natural ventilation occurs when desired air flows occur through windows, doors, chimneys and other building openings. Mechanical ventilation is the mechanically induced movement of air through the building. Mechanical systems usually condition and filter the air and allow for the entry of outdoor air through outdoor dampers. Infiltration is the unwanted movement of air through cracks and openings into the building shell.

The outside air ventilation rate of a building affects indoor air quality. It determines the extent to which contaminants are diluted and removed from the indoor environment. The extent to which outside air ventilation is effective in diluting indoor contaminants depends on how well outside air is mixed with indoor air and is reflected by ventilation efficiency. Ventilation efficiency can be reduced by air short-circuiting from the supply diffusers to the return inlets, by modular furniture partitions, and differences between the supply air temperature and the room air temperature.

The rate at which outside air is supplied to a building is specified by the building code at the design stage. Outside air ventilation rates are based primarily on the need to control odors and carbon dioxide levels (e.g., occupant-generated contaminants or bioeffluents). Carbon dioxide is a component of outdoor air whose excessive accumulation indoors can indicate inadequate ventilation.

Lack of adequate ventilation contributes to indoor air related health complaints. Specific deficiencies that produce air quality problems include inadequate outside air supply, poor air distribution, poor air mixing (and therefore poor ventilation efficiency), inadequate control of humidity, insufficient maintenance of the ventilation system, inadequate HVAC system capacity and inadequate exhaust from occupied areas. Inadequate outdoor air supply and distribution and insufficient control of thermal conditions can result from strategies to control energy consumption. In approximately 500 indoor air quality investigations conducted in the late 1970's and early 1980's, the National Institute for Occupational Safety and Health (NIOSH) found that the primary causes of indoor air quality problems were inadequate ventilation (52%), contamination from outside the building (10%), microbial contamination (5%), contamination from building fabric (4%) and unknown sources (13%) [56 FR 47892]. To date, NIOSH has conducted over 1,100 IAQ related investigations, but has not yet evaluated them to provide updated estimates.

OSHA, therefore, believes that it is necessary to require maintenance of the HVAC system components that directly affect IAQ, since failure to do so results in the degradation of IAQ. Standards of HVAC maintenance vary and sometimes are deficient where untrained personnel are designated to maintain complex systems. It is, also, customary for companies to defer maintenance for economic and budgetary reasons, with adverse impacts on IAQ. Some examples of maintenance deficiencies include: plugged drains on cooling coil condensate drip pans (resulting in microbial contamination); failed exhaust fans in underground parking garages; microbial fouling of cooling tower water from lack of water treatment with biocides resulting in legionellosis cases; and failure of the automatic temperature control system resulting in lack of outside ventilation air.

4. Building Characteristics

During the last 25 years, technical and socioeconomic changes have profoundly influenced the methods employed to plan, design, construct and operate buildings. Buildings system design, maintenance and operation can, and regularly do, provide acceptable indoor environments. However, neglect or disregard of the sources of indoor air contaminants, or of the proper design, operation and maintenance of building system components which influence indoor air quality can create an uncomfortable and unhealthy indoor atmosphere [Ex. 3–1075H2].

The oil embargo of 1973 brought about the realization that considerable savings could be made in reducing the consumption of energy used to heat and cool buildings. Prior to 1973, the energy to heat and cool buildings was much cheaper and the buildings reflected that reality. Building enclosures had lower insulating values and allowed more infiltration. More air was circulated to the occupied spaces and more outdoor air was provided for ventilation. This resulted in a lower concentration of pollutants and higher velocities of air motion in indoor air. Office buildings were divided into individual rooms with their own walls as opposed to the current practice of open spaces with movable screens [Ex. 4–74].

The centralization of services and the expanding economy have led to concentration of office space in the cities. The cost of land has shaped buildings into high-rise structures. The cost of materials and popularity of mirror glass has led to the sprouting of hundreds of what may be termed "glass boxes". These boxes are sealed to keep out noise and pollution—mainly from traffic.

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include adhesives, carpeting, caulks, glazing compounds, and paints [Ex. 4–33]. These materials, as well as furnishings can act as a sponge or sink in which VOCs are absorbed and then re-emitted later.

Appliances, office equipment, and supplies can emit VOCs and also particulates [Ex. 4–33]. Table III–2 lists the many contaminants that can be emitted from these point sources. There is an indirect relationship between the age of the point source and the potential rate of contaminant emission [Ex. 4–33].

TABLE III–1.—EMISSIONS FROM BUILDING MATERIALS OR INTERIOR FURNISHINGS

Material	Typical pollutants emitted
Adhesives	Alcohols. Amines. Benzene. Decane. Dimethylbenzene. Formaldehyde. Terpenes. Toluene. Xylenes.
Caulking Compounds	Alcohols. Alkanes. Amines. Benzene. Diethylbenzene. Formaldehyde. Methylethylketone. Xylenes.
Carpeting	Alcohols. Formaldehyde. 4-Methylethyl- benzene. 4-Phenylcyclohexene. Styrene.
Ceiling Tiles	Formaldehyde.
Clipboard/Particle Board.	Alcohols. Alkanes. Amines. Benzene. 3-Carene. Formaldehyde. Terpenes. Toluene.
Floor and Wall Coverings.	Acetates. Alcohols. Alkanes. Amines. Benzenes. Formaldehyde. Methyl styrene. Xylenes.
Paints, Stains & Varnishes.	Acetates. Acrylates. Alcohols. Alkanes. Amines. Benzenes. Formaldehyde. Limonene. Polyurethane. Toluene.

TABLE III–2.—EMISSIONS FROM APPLIANCES, OFFICE EQUIPMENT AND SUPPLIES [1]

Appliances	Carbon Monoxide. Nitrogen Dioxide. Sulfur Dioxide. Polyaromatic hydrocarbons.
Carbonless Copy Paper.	Chlorobiphenyl. Cyclohexane. Dibutylphthalate. Formaldehyde.
Computers/Video Display Terminals.	n-Butanol. 2-Butanole. 2-Butoxyethanol. Butyl-2-Methylpropyl phthalate.
Computer/Video Display Terminals.	Caprolactam. Cresol. Diisooctyl phthalate. Dodecamethyl cyclosiloxane. 2-Ethoxyethyl acetate. Ethylbenzene. Hexanedioic acid. 3-Methylene-2-pentanone. Ozone. Phenol. Phosphoric Acid. Toluene. Xylene.
Duplicating Machines	Ethanol. Methanol. 1,1,1-Trichloroethane. Trichloroethylene.
Electrophotographic Printers, Photocopiers & Related Supplies.	Ammonia. Benzaldehyde. Benzene. Butyl methacrylate. Carbon black. Cyclotrisiloxane. Ethylbenzene. Isopropanol. Methylmethacrylate. Nonanal. Ozone. Styrene. Terpene. Toluene. 1,1,1-Trichloroethane. Trichloroethylene. Xylenes. Zinc stearate combustion Products.
Microfiche Developers/Blueprint Machines.	Ammonia.

TABLE III–2.—EMISSIONS FROM APPLIANCES, OFFICE EQUIPMENT AND SUPPLIES [1]—Continued

Preprinted Paper Forms.	Acetaldehyde. Acetic Acid. Acetone. Acrolein. Benzaldehyde. Butanal. 1,5-Dimethylcyclopentene. 2-Ethyl furan. Heptane. Hexamethyl cyclosiloxane. Hexanal. 4-Hydroxy-4-methyl pentanone. Isopropanol. Paper dust. Propionaldehyde. 1,1,1-Trichloroethane.
Typewriter Corrections Fluid.	Acetone. 1,1,1-Trichloroethane.

[1] Source: [Ex. 4–33]

Emissions from equipment, such as computers, will decrease over time compared to emissions from equipment that continually use chemicals. Emissions from such equipment (e.g., laser printers) that use chemicals continually, will obtain a steady state concentration dependent upon the chemicals used and frequency of equipment use.

B. Microbial Contamination

Three conditions must exist in buildings before microbial contamination can occur: high humidity (over 60%), appropriate temperatures (varies according to microbe), and appropriate growth media [Exs. 3–61, 4–33]. These conditions are found in heating, ventilating, and air conditioning (HVAC) systems. HVAC systems provide multiple sites for microbes to grow (reservoir) and also the means to disperse the microbes throughout the ventilated space. These reservoirs of microbial growth, if allowed to proliferate unchecked, can lead to indoor air quality problems once the microbes or microbe-related products, such as endotoxins, are dispersed.

Building materials that have been soaked with water, such as fiberglass insulation in air handlers, furnishings and fabrics, ceiling tiles, and carpeting are excellent media for microbial growth. Biological organisms, including fungal spores, bacteria, viruses, pollens, and protozoa derived from mold growth have been identified in humidifiers with stagnant water, water damaged surfaces and materials, condensing coils and

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natural ventilation. The study included 880 workers. Mendell estimated relative risks for several building related symptoms and a subset of these estimates are shown in Table IV–11. In an effort to define comparable symptoms between the reported national statistics from NHIS and Mendell's study and for computational ease OSHA grouped "runny nose", "stuffy nose", "dry/irritated throat", and "dry/irritated/itching eyes" as upper respiratory/mucous membrane symptoms. Mendell reported relative risks for upper respiratory conditions and frequent headaches in air-conditioned buildings as compared to naturally ventilated buildings. The relative risk for frequent headaches was reported to be 1.5. For upper respiratory conditions, such as "stuffy nose", "runny nose", etc., the relative risks ranged from 1.4 to 1.8. OSHA used 1.4 as an observed relative risk for upper respiratory conditions.

CDC reports in the "Current Estimates from the National Health Interview Survey, 1992" that the annual rate for severe headaches, requiring medical attention or activity restriction, is at least 5 per thousand and the rate for upper respiratory conditions is at least 9 per thousand. In addition, it is estimated that the proportion of office buildings in the U.S. with air-conditioning is 70 percent (see Preliminary Regulatory Impact Analysis section). Using the above information and the same methodology as described in section IV-D, OSHA estimated that the lifetime excess burden for severe headaches experienced in air-conditioned office buildings is 57 per one thousand exposed employees and the lifetime risk for acute upper respiratory conditions is 85 per one thousand exposed employees. OSHA's risk estimates for indoor air are shown in Table IV–12. OSHA used data derived from a study of air-conditioned office buildings to make an assessment of the occupational risk in all air-conditioned buildings. Furthermore, OSHA made an implicit assumption that an increase in work-related headaches associated with an air-conditioned office environment occurs in the same proportion as headaches which can be severe enough to affect work activity. OSHA seeks comment on the applicability of the Mendell study for estimating occupational risk in air-conditioned buildings due to poor indoor air quality. In addition, OSHA seeks comment on its methodology of developing annual and lifetime risk estimates attributable to occupational exposures.

TABLE IV–11.—CALIFORNIA HEALTHY BUILDING STUDY COMPARING BUILDINGS WITH NATURAL VENTILATION TO BUILDINGS WITH AIR-CONDITIONING [1]

Health outcome	Relative risk	Confidence interval
Upper respiratory symptoms:		
Runny nose	1.5	(0.9–2.5)
Stuffy nose	1.8	(1.2–3.7)
Dry/irritated throat	1.6	(0.9–2.7)
Dry/irritated/itchy eyes	1.4	(0.9–2.2)
Frequent headaches	1.5	(0.9–0.3)

[1] Study subjects were asked whether the symptoms were occurring often or always at work and improving when away from work.

TABLE IV–12.—OSHA'S ESTIMATES OF RISK FOR WORKERS IN AIR-CONDITIONED BUILDINGS [1]

	Annual risk [2]	Lifetime occupational risk [3]
Severe headaches [4]	1.296	57
Upper respiratory symptoms [5]	1.969	85

[1] Risks are expressed as number of cases per 1,000 workers at risk.

[2] The annual risk is estimated assuming that the prevalence of air-conditioned office buildings in the U.S. is 70 percent.

[3] Assumes 45 years of occupational exposure and is calculated as $1-(1-p)^{45}$, where p is the annual risk.

[4] Defined as headaches that either require medical attention or restrict activity.

[5] Defined as runny nose, stuffy nose, dry/irritated throat and dry/irritated/itchy eyes and being severe enough to either require medical attention or restrict activity.

F. Pharmacokinetic Modeling of ETS Exposure

In developing a final rule, OSHA would like to consider the use of a physiologically based pharmacokinetic (PBPK) model in an effort to develop a clear and complete picture of factors that may affect environmental exposure measurements, internal dose estimates and ultimately estimates of expected risk attributed to ETS exposure at the workplace. OSHA is seeking comment on appropriate methodology, available data, etc. The following discussion offers an explanation of OSHA's approach to this issue and an opportunity for the Agency to solicit comment on specific points of concern as they relate to the use of pharmacokinetics in estimating occupational risk from exposure to ETS.

Estimating the risk from exposure to ETS requires the use of some measure of the extent of exposure. Possible measures, or metrics, can range from categorical ranking based on survey responses to direct measurement of ETS-related chemicals in the body fluids of exposed individuals. In general, the use of an internal measure of individual exposure would be preferred over measurements of environmental contamination, such as airborne chemical or particulate concentrations. In particular, considerable attention has been given in the scientific literature to the possible use of cotinine concentrations in body fluids as a biomarker of ETS exposure [Exs. 4–24, 4–146, 4–165, 4–263, 4–316]. However, obtaining a dependable estimate of exposure from measurements of a chemical's concentration in body fluids requires a quantitative understanding of the chemical's pharmacokinetics; its uptake, distribution, metabolism, and excretion. Following is a review of the evidence concerning the suitability of cotinine as an internal biomarker for ETS exposure.

1. Considerations for Selection of a Biomarker for ETS

A biomarker should, to the greatest extent possible, accurately represent the individual's exposure to the substance of concern and have relevance to a specific endpoint. In the case of ETS, there are several relevant endpoints, with principal attention being given to heart disease and lung cancer. Each different endpoint may be mediated by a different subset of the components of ETS, and therefore the appropriate biomarker(s) for each endpoint could be different.

2. Cardiovascular Effects

Cardiovascular effects resulting from exposure to ETS have been associated with carbon monoxide (CO), nicotine, and more recently with polycyclic aromatic hydrocarbons (PAHs) [Ex. 4–123]. Each of these is associated with a different fraction of ETS; CO is a gas phase constituent, nicotine is a low volatility vapor, and PAHs are absorbed on particulates. Because of the significant differences in physical fate and transport, a strategy for the use of biomarkers for cardiovascular effects of ETS would ideally make use of separate markers for CO, nicotine, and PAHs.

The most common internal measure of CO exposure is blood carboxyhemoglobin (HbCO). Blood HbCO provides a useful measure of exposure to CO, and can be related to the cardiovascular effects of CO. A way to determine the occupational

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the breathing zone of nearby employees. The carcinogenicity of ETS discounts the use of general ventilation as an engineering control for this contaminant.

The major ventilation guidance document available to HVAC practioners (e.g., designers, maintenance, and operators), is Standard 62–1989 titled "Ventilation for Acceptable Indoor Air Quality" [Ex. 4–333]. The standard is published by the American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc. (ASHRAE) and it specifies recommended minimum design outside air ventilation rates for 91 different applications. Based on this current ventilation standard, a typical commercial HVAC system serving general office space should prescriptively deliver 20 cubic feet per minute per person (cfm/person) of outside air to the occupied space to dilute occupant generated contaminants like carbon dioxide (CO_2) and body odors. This ventilation rate would provide what ASHRAE defines as "acceptable indoor air quality" (e.g., sensory comfort) to satisfy at least 80% of the building occupants. The prescribed ventilation rates in ASHRAE Standard 62–1989 are proportional to the occupants in the space (e.g., cfm/PER PERSON) because of the presumption that the contamination produced is in proportion to the occupant density.

The foreword of ASHRAE Standard 62–1989 states "with respect to tobacco smoke and other contaminants, this standard does not, and cannot, ensure the avoidance of all possible adverse health effects, but it reflects recognized consensus criteria and guidance." As published, ASHRAE Standard 62–1989 did not include any summary and/or explanation documentation which would explain the basis of the consensus standard. Without this documentation, it can only be inferred that the standard was mostly based on satisfaction of sensory comfort rather than based on the control of contaminants like ETS which may contribute to adverse health effects like lung cancer and heart disease.

The method of room air distribution found in most HVAC systems is a mixing system that attempts to create an environment of uniform air velocities, temperatures and humidities in the occupied zone of a room (e.g.; floor to 6 feet above floor). In this occupied zone, air velocities less than 50 feet per minute (fpm) and minimization of temperature gradients will promote occupant comfort. In a conventional mixing system where the supply air diffusers (outlets) and the return air grilles are both located in the ceiling, the air motion in the occupied zone could be characterized as "gentle drift" toward the ceiling where the room air is then mixed with the conditioned air being delivered to the room through the supply air diffusers [1993 ASHRAE Handbook, Ch.31]. Because of natural convection currents and thermal buoyancy forces it is common, especially during heating season, to have stagnant zones. In a mixing room air distribution system, the emphasis is on comfort.

There are other room air distribution schemes which consider contaminant control and have been used in the industrial environment like displacement ventilation and unidirectional (plug-flow) airflow ventilation. In these schemes, there is an attempt to move contaminants directionally along a clean to less clean gradient. These schemes are seldom used in conventional HVAC systems due to their cost, feasibility and compromise of comfort issues.

From the industrial hygiene perspective, local exhaust ventilation, specific to each source, would be the preferred and recommended method for controlling occupational exposures to contaminant point sources like ETS. Such specific ventilation is effective because the contaminant is captured or contained at its source before it is dispersed into the work environment where only ineffective general dilution ventilation is available to control exposures.

A designated smoking area which is enclosed, exhausted directly to the outside, and maintained under negative pressure is sufficient to contain tobacco smoke within the designated area. Such areas could be considered an application of local exhaust ventilation because the contaminant is being exhausted from a confined source without dispersal into the general workspace.

IV. Preliminary Quantitative Risk Assessment

A. Introduction

The determining factor in the decision to perform a quantitative risk assessment is the availability of suitable data for use in such an assessment. A wide spectrum of health effects have been associated with exposure to indoor air pollutants and ETS. These effects range from acute irritant effects to cancer. In the case of ETS, OSHA has determined that data are available to quantify two types of risk: lung cancer and heart disease. For this risk assessment, OSHA defines "heart disease" to be coronary heart disease excluding strokes, as defined in the Framingham study [Ex. 4–108]. In the case of indoor air pollutants, the only data available to OSHA were on specific acute health effects, such as severe headaches, excluding migraines, and other respiratory conditions, such as "stuffy nose", "runny nose", etc. OSHA is aware that there are more serious conditions such as legionellosis and hypersensitivity diseases associated with poor indoor air and suspected to be potential occupational hazards. However, the Agency currently does not have adequate data to conduct a quantitative risk assessment addressing these risks in the workplace. OSHA is continuing to develop appropriate methodology to address risk estimations for conditions related to poor indoor air quality in the workplace and is requesting input on data sources relevant to these efforts.

There is uncertainty associated with the quantification of any kind of risk. In this risk assessment, OSHA has tried to describe many of the sources of uncertainty and to address their implications for OSHA's estimates of risk.

For the purpose of this rulemaking and for deriving a quantitative estimate of occupational risk, OSHA has concentrated on information and data concerning heart disease and lung cancer as potential effects associated with exposure to ETS.

B. Review of Epidemiologic Studies and Published Risk Estimates

As a first step in this risk assessment, OSHA critically reviewed epidemiologic studies associating exposure to ETS or indoor air pollutants with adverse health effects. The purpose of such a critical evaluation was to determine whether exposure to ETS is a causal factor in cancer and heart disease and whether exposure to indoor air pollutants has caused a significant increase in acute irritant effects. The critical review also enables OSHA to select those studies that have potential for use in a quantitative risk assessment. Tables IV–1 and IV–2 contain a summary of OSHA's assessment of several epidemiologic studies of ETS exposed individuals.

OSHA evaluated studies on exposure to ETS to determine the importance and weight of each study in the overall hazard identification process. Of those, it was determined that fourteen showed a statistically strong association between exposure to ETS and lung cancer and four showed a significant association between ETS exposure and heart

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workplace is analyzed further it becomes apparent that the overwhelming exposure location for these employed nonsmokers is the workplace (Table III–9). As indicated in Table III–9, 77% of males and 85% of females were exposed an average of 313 minutes and 350 minutes, respectively.

One other finding is that the more time spent at work, the higher the likelihood of greater ETS exposure. For example, the average duration of exposure to homemakers was approximately 2 hours a day, for workers the average duration of exposure was approximately 3 hours a day.

TABLE III–8.—PERCENTAGE OF EMPLOYED NONSMOKERS EXPOSED TO ETS AND AVERAGE MINUTES OF EXPOSURE (IN PARENTHESES) [1]

Exposure location	Males	Females	Total
Home	9 (134)	13 (109)	11 (123)
Work	51 (313)	38 (350)	46 (324)
Other indoor	28 (89)	35 (77)	31 (85)
Outdoor	12 (118)	14 (79)	13 (104)

[1] Source: [Ex. 4–169].

TABLE III–9.—PERCENTAGE OF EMPLOYED NONSMOKERS EXPOSED TO ETS AND AVERAGE MINUTES OF EXPOSURE (IN PARENTHESES) OF THOSE WHO REPORTED ETS EXPOSURE AT WORK [1]

Exposure location	Males	Females	Total
Home	1 (147)	2 (180)	2 (158)
Work	77 (313)	85 (350)	80 (324)
Other indoor	15 (92)	9 (102)	13 (94)
Outdoor	6 (176)	4 (140)	5 (166)

[1] Source: [Ex. 4–169].

Work breaks and meals at work were the work activities most closely associated with ETS exposure, 51% and 35% respectively versus 27% for work per se [Ex. 4–271]. In other words, nonsmokers experienced ETS exposure in break areas more than in general work areas.

When white collar versus blue collar workplaces were compared, 37% of factories/plants versus 22% of offices had episodes of ETS exposure, suggesting that blue collar nonsmoking workers have a greater exposure to ETS than white collar workers. For the CAP population, twice as many workers were employed in offices as were in factories [Ex. 4–271]. The most ETS exposed nonsmokers were those with 10 or more hours per day of work [especially at plants/factories], more than 2 hours per day of restaurant time, and more than 1 hour per day of bar or nightclub time.

Robinson et al. [Ex. 4–271] concluded that the probability of passive smoking is highest for a combination of various social and work activities, consistent with the notion that activities that involve more people involve a greater chance of contact with people who smoke. A limitation of the CAP survey is that the data do not provide information on the intensity of exposure in the various microenvironments [Ex. 4–271].

In summary, the CAP study showed that the most powerful predictor of potential exposure to ETS was being employed. Respondents who spent more than ten hours a day at the workplace were found to report more ETS exposure than those working less than 10 hours a day or not at all. Further data from this study show that the workplace is the location with the highest reported exposure to ETS in enclosed environments, and such exposure is on average nearly three times more prevalent at work than at home.

Another relevant data source for assessing ETS exposure in the workplace is the National Health Interview Survey (NHIS) conducted by the Centers for Disease Control and Prevention (CDC). In its Health Promotion and Disease Prevention (NHIS-HPDP) supplement, CDC collected self-reported information on smoking from a representative sample of the U.S. population [Ex. 4–51]. The results suggest that at least 19% of employed nonsmokers experience ETS exposure at work. The CDC study results represent the prevalence of occupational exposure among nonsmoking adults [see section IV for further discussion of this study].

In a smaller study, Cummings et al. [Ex. 4–67] studied the prevalence of exposure to ETS in 663 (44% male) never- and exsmokers aged 18–84 years, who attended a cancer clinic in Buffalo, New York in 1986 (see Table IV–9). The study employed questionnaires and analysis of urinary cotinine levels. The subjects were asked if they were exposed to passive smoke either at home or at work in the four days preceding the interview. A further analysis of this data focusing on workers from this survey determined that overall, 339 subjects were currently employed. Of these 264 (77%) reported ETS exposure at work. The percentage of subjects exposed to ETS at both work and the home was 29% (n=99). The percentage of subjects exposed at home, but not at work was 7% (n=23). The percentage of subjects exposed at work, but not at home was 49% (n=165). The percentage of subjects exposed neither at home or work was 15% (n=52). This further analysis indicates that the workplace is a significant source of ETS exposure for nonsmoking, employed people.

Emmons et al. [Ex. 4–98] reported on a study of 186 nonsmoking volunteers from workplace settings selected to have a wide range of exposure to ETS. The subjects were asked to keep a 7-day exposure diary. The worksites ranged from those with minimal restrictions and high levels of exposure (long-term care and psychiatric facilities, chemical dependency and treatment centers, and a VA Hospital) to those with extensive restrictions and low exposure (e.g., state health department and community hospitals). Seventy-six percent of the subjects reported being regularly exposed to ETS in the workplace. The percentage of subjects reporting exposure at work is similar to that found by Cummings et al. [Ex. 4–67]. Nonsmokers encountered significantly more exposure to ETS at work (50%) as

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E. Technological Feasibility and Compliance Costs

This section presents OSHA's preliminary compliance cost estimates for the proposed standard on indoor air quality. The cost analysis covers the major proposed provisions for which data are available.

OSHA requests more information on the consideration for the relationship of employers and facility owners. The decision to implement any IAQ improvements will be greatly influenced by the relationship between employers and landlords. Since changes in building ventilation systems will be made by landlords, employers may have to negotiate agreements to ensure that they can meet the OSHA standard. On the requirement for ETS, landlords in turn are likely to pressure employers to ban smoking; thereby, forestalling any need for construction of designated smoking rooms. This section also examines the technological feasibility of complying with proposed regulation.

1. Technological Feasibility

As interpreted in the Benzene and Cotton Dust cases, the Occupational Safety and Health Act of 1970 requires that the Agency, with regard to exposure to toxic substances, is to reduce significant risk of material health impairment to the extent feasible. Accordingly, as part of the investigation of the potential effects of the OSHA proposal, OSHA has examined both the technological and economic feasibility of the proposal. The economic feasibility assessment appears later.

OSHA's assessment of the technological feasibility is based on an examination of what would be required to comply with the proposal, along with a review of existing practices among affected establishments. With regard to this proposal, problems with technological feasibility, by and large, are not evident. Employers are required to operate their HVAC systems within those parameters originally designated for the equipment. While many employers may choose to provide separately ventilated smoking areas, this is an option, not a requirement, under the proposed regulation. This technology is widespread currently and can be used to achieve compliance with the proposed standard.

For example, in some situations, such as hotels and prisons, employees have as their workplace the residence of others who live in that building. Restaurants, bars and other "public" places expose employees to customer's tobacco smoke. While it is technologically feasible to ban smoking in those establishments, there may be other problems, legal and economic. While it is theoretically possible to minimize employee exposure to ETS in such a work environment through special ventilation, in the absence of modified customer service arrangements, actually eliminating worker exposure to ETS would likely prove difficult. Consequently, the selection process for one of the smoking policy alternatives for a particular workplace must consider both the physical limitations of the building or firm and the building's use. In addition, some employers may be using their building facilities for purposes for which the original design did not intend, and for which retrofitting might prove difficult. OSHA requests comment on those workplaces for which compliance with the proposed standard would prove technologically challenging. OSHA will consider additional information on the ability of firms to implement IAQ programs.

2. Compliance Costs

OSHA estimated preliminary costs of complying with the proposed standard. OSHA's cost assumptions and methodologies are based on information available from the rulemaking record. Further detailed industry analysis will be developed by the Agency.

Table VI–8 contains OSHA's estimates of the annualized first-year and the annual recurring costs of full compliance with the proposed rule. The annualized first-year cost of compliance is $1.4 billion. The cost for eliminating exposure to ETS may range from $0 to $68 million depending on whether establishments shall ban smoking or allow smoking in designated areas. OSHA estimated that the annual cost of compliance with the IAQ standard will be $8.1 billion, of which the most costly provision will be for the building systems operation and maintenance, $8.0 billion.

OSHA developed cost estimates for the affected industries using the following categories of information: (1) Provisions of the proposed standard requiring activities; (2) the number of potentially affected buildings, establishments and employees; (3) the percentage of establishments or buildings in each industry currently in compliance with each proposed requirement; and (4) the unit costs for bringing establishments into compliance with the various provisions of the proposed standard. These four items were combined to produce OSHA's estimated costs of compliance.

Costs were estimated on an annual basis, with total annual costs calculated as the sum of annualized initial costs and annual recurring costs. All capital costs and non-recurring first year costs were annualized over the service life of the equipment or administrative activity, at a discount rate of 10 percent.

(a) Developing Indoor Air Quality Compliance Programs. The proposed standard requires establishments to prepare written operations plans which would describe information required for the daily operation and management of the building systems [9] and maintenance. The plan should provide an overview of the building and system, using a short text description and single-line schematics or as-built construction documents. The operations information would also describe how to operate the HVAC systems so that it performs with the reported design criteria. In addition, the operations information should include: (1) Special procedures like seasonal start-ups and shutdowns, and (2) a list of operating performance criteria such as minimum outside air ventilation rates, potable hot water storage and delivery temperatures, range of space relative humidities and any space pressurization requirements, (3) an evaluation of the need to retrofit the HVAC system when the design occupancy levels are exceeded, and (4) a checklist for visual inspection of building systems.

[9] Building systems include but are not limited to the heating and air conditioning (HVAC) system, the potable water systems, the energy management system and all other systems in a facility which may impact IAQ.

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Buildings designed after 1973 have incorporated many energy conservation measures that range from adjusting thermal comfort zones to increased awareness of lighting efficiency, to designing new operating methods for "sealed building" [Ex.3–1159, p.1]. In large buildings, outside air ventilation rates were also reduced by closing outside air dampers in mechanical ventilation systems at nights, on weekends and sometimes even during occupancy. As a result of these measures, which primarily reduced costs for conditioning outdoor air as opposed to increasing energy efficiency, considerable energy savings have been achieved in buildings.

In addition, during the 1970's variable air volume (VAV) HVAC systems became widely accepted. VAV systems condition supply air to a constant temperature and insure thermal comfort by varying the airflow. Early VAV systems did not allow control of the outside air quantity, so that a decreasing amount of outside air was provided as the flow of supply air was reduced.

In some cases, building design flaws contribute to the poor quality of indoor air, such as locating air intake vents near to a loading dock or parking garage. Design flaws of interior space also contribute to indoor air problems. Most building cooling systems are designed to remove the heat generated by office machines, employees and light. The heat generated by these sources often exceeds the capacity of the HVAC system to remove it [Ex.3–1159C1]. Ideally with effective filtration and management systems, the air indoors should be cleaner than the air outdoors.

5. Profile of Affected Buildings

Estimates of the number of buildings potentially affected by the indoor air standard were developed by OSHA based on Department of Energy's commercial building energy consumption survey (CBEC) 1989[7] [Ex. 4–303]. There is a total of 4.5 million commercial buildings in the United States. Commercial buildings are defined as all non-manufacturing/ industrial and non-residential structures. Table VI–1 presents the distribution of buildings by use, occupancy and thermal conditioning. Approximately 28 percent of all buildings are for mercantile or services. Other uses include offices (15 percent), assembly and warehouses (14 percent each), food service (5 percent), lodging (3 percent) and food sales and healthcare (2 percent each). The "other" category (1 percent) covers buildings such as public restrooms and buildings that are 50 percent or more commercial but whose principal activity is agricultural, industrial/manufacturing or residential.

On average, the largest types of buildings are for education and health care. Mercantile and service buildings account for the greatest number and floorspace of any single activity category. Office buildings account for nearly as much floorspace, but far fewer buildings. Together office and mercantile buildings represent almost 40 percent of all buildings and floorspace. Warehouses and assembly buildings both are almost as numerous as office buildings, but account for less floorspace. Over 62 percent of buildings have only one floor and 13 percent have three or more floors. Most buildings (69%) house single establishments. Government occupied buildings represent 13 percent.

TABLE VI–1.—EMPLOYEES WORKING IN BUILDINGS AND OTHER BUILDING CHARACTERISTICS

Principle building activity	Number of buildings	Percent of all buildings	Total number employees
Principal building activity:			
Assembly	615,000	14	4,012,000
Education	284,000	6	7,204,000
Food sales	102,000	2	844,000
Food service	241,000	5	1,943,000
Health care	80,000	2	4,225,000
Lodging	140,000	3	3,092,000
Mercantile and service	1,278,000	28	12,414,000
Office	679,000	15	27,780,000
Parking garage	45,000	1	332,000
Public order and safety	50,000	1	861,000
Warehouse	618,000	14	4,377,000
Other	62,000	1	2,111,000
Vacant[1]	333,000	7	1,472,000
Total	4,527,000		70,667,000
Building occupants:			
Single establishments—owner occupied	2,445,000	54	
Multiple establishments—owner occupied	369,000	8	
Single establishments—nonowner occupied	672,000	15	
Multiple establishments—nonowner occupied	259,000	6	
Vacant	206,000	5	
Government buildings	577,000	13	
Thermal conditioning:			
Heated	3,865,000	85	
Entire building	2,739,000	60	
Part of building	1,126,000	25	
Cooled	3,184,000	70	
Entire building	1,550,000	34	

[7] The commercial building and energy consumption survey is a triennial national sample survey of commercial buildings and their energy suppliers. This survey is the only source of national level-data on both commercial building characteristics and energy consumption.

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Source: https://www.industrydocuments.ucsf.edu/docs/pxhl0000

[2] Initial costs are annualized over 10 years at a 10 percent interest rate. Training for maintenance workers is estimated to take one—half hour. Compensation wage rate is $10.95 per hour. Cost includes an additional 7.5 minutes per employee to cover trainer cost. Total training cost per employee is $6.84. NA: Data not available.

Source: OSHA, Office of Regulatory Analysis, 1994.

Establishments in large high rise buildings may also find it desirable to provide such rooms to facilitate break periods. Consequently, in order to reflect the degree to which establishments will provide separate smoking areas, OSHA developed some estimates based on the characteristics of the stock of buildings and the percentage of companies currently banning smoking in the workplace.

OSHA has no data on the number of establishments currently permitting smoking in designated smoking areas. OSHA estimated that 50 percent of large establishments with floor space greater than 100,000 square feet and with more than three floors will provide designated smoking areas. OSHA also assumed that 50 percent of all eating and drinking places and hotels and other lodging places may provide separate designated smoking areas. For these establishments, OSHA then applied the percentage of companies that will ban smoking based on the rates provided from a survey conducted by the Administrative Management Society Foundation (AMS) on current practices for smoking policies in the workplace. According to the survey, 25 percent of the companies completely ban smoking on their premises. However, the percentages varied by SIC as follows: manufacturing (23%), transportation and utilities (36%), banking and finance (28%), insurance (38%), retail and wholesale (7%), and services (18%). Also, 72 percent felt that smoking in the workplace should be either banned or restricted [H–030—Ex. 75].

Firms opting to make available designated smoking areas are expected to incur initial capital costs. OSHA assumed that in many cases existing rooms or offices can be converted into a designated smoking area. Average cost estimates for retrofitting the HVAC system ranges from $4,000 for a 150 square feet room (which could accommodate up to 10 smokers) [Ex. 4–265] to $25,000 for 1,000 square feet (which could accommodate 30 to 65 smokers) [Ex. 3–643]. The HVAC retrofit represented in these estimates typically includes: (1) blocking off the return air inlet from the room, (2) providing a transfer air path, and (3) providing an exhaust fan and exhaust air pathway to the outside. The exhaust fan capacity would exceed air supplied to the room in sufficient quantity to create a negative pressure in the smoking room relative to surrounding areas to ensure containment of the contaminant. In order to achieve negative pressure some architectural modifications may be necessary to provide a tight enclosure. OSHA did not estimate an additional cost for housekeeping since such activities would have been performed prior to the promulgation of the proposed standard.

Most facilities exhaust air from toilet rooms and also relieve air brought in for ventilation and economizer cooling [10(a)]. The amount of exhaust air from a designated smoking area is inconsequential compared with the quantities of air leaving the building through toilet room exhaust and relief. Therefore, OSHA did not include recurring cost for the provision of a separately ventilated smoking area.

The equation for determining cost for allowing smoking in designated areas is as follows:

$$C_t = (N_e \times (1-P_s) + N_d \times (1-P_{sm}))\ P_c \times C_r$$

where

C_t = cost for providing designated areas

N_e = number of establishments in buildings with 3 or more floors and floorspace greater than 100,000 sq.ft.

P_s = percentage of establishments banning smoking

N_d = 50 percent of establishments in Eating and Drinking Places (SIC 58), and Hotel (SIC 70)

P_{sm} = percentage of establishments in SIC 58 and SIC 70 banning smoking[c].

P_c = percentage of establishments providing designated smoking areas (50%)

C_r = cost for setting up a separate smoking area ($4,000 for a 150 sq.ft. room that accommodates up to 10 smokers, furnishings existing)

Initial costs are annualized over 20 years at 10 percent interest rate. As presented in Table VI–12, the total annual cost is estimated at $68 million. OSHA did not include a cost estimate for the government sector at this time.

(f) Air Quality during Renovation and Remodeling. The proposed standard requires that during renovation and remodeling appropriate controls are utilized to minimize degradation of the indoor air quality of employees performing such activities and employees in other areas of the building. The basic characteristics of available control practices include: ventilation system/high efficiency particulate air (HEPA) vacuum; regulated areas, isolation or containment of work areas and appropriate negative pressure containment; outside air intakes, return/recirculation air streams or plenums; notification of employees and contractors.

For buildings with asbestos presence, the control practices under the OSHA asbestos standard are current industry practice. A survey developed for obtaining information on practices to control exposure to asbestos in buildings shows that asbestos-related work represents 16 percent of renovation activities whereas general remodeling is 61 percent and major repair and maintenance are 12 percent [Ex. 4–64].

TABLE VI–12.—OPTIONAL COST FOR PROVIDING SEPARATE SMOKING AREAS

	Number of establishments providing designated smoking areas [1]		Annualized first-year cost [2] ($ million)
	In single establishment buildings	In multi-establishment buildings	
Agriculture, forestry, fishing	43	8	$0.024
Mining	4	1	0.002
Construction	105	21	0.059
Manufacturing	65	13	0.037

[10(a)] Use of outside air for cooling—"free cooling".

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VII. Summary and Explanation

The requirements set forth in this notice are those which, based on currently available data, OSHA believes are necessary and appropriate to control conditions which may degrade indoor air and pose a significant risk of material impairment to employees in their work environments. The Agency considers that a broad approach to the control of IAQ problems, as proposed in this notice, will most effectively lead to a reduction in associated risk to employees [Exs. 3–2, 3–26, 3–37, 3–41, 3–239, 3–287, 3–434, 3–500, 3–502]. OSHA has considered all data submitted in response to the Request for Information, as well as other scientific data which has been made a part of the record in this proceeding in arriving at these proposed provisions regarding regulation of indoor air quality.

The following sections provide a summary of each provision of the proposal and a statement of their intent and purpose. Exhibit numbers included in this Summary and Explanation are citations to supporting comments and data submitted to the record in response to the RFI.

The Agency solicits data, views, and comments on all provisions proposed in this notice. OSHA is interested in whether or not the proposed provisions are necessary, appropriate, and adequate to achieve the goals of the standard and why. Interested persons should also comment on whether or not the proposed provisions are technologically and economically feasible and why, and whether additional or alternative provisions addressing indoor air quality should be included in the standard and why.

Scope and Application: Paragraph (a)

OSHA is proposing that these standards cover all employees under its jurisdiction, including employees in general industry, shipyards, longshoring, marine terminals, construction, and agriculture. To accomplish this, OSHA is proposing to publish an identical complete standard for general industry at 29 CFR 1910.1033, for shipyards at 29 CFR 1915.1033, and for construction at 29 CFR 1926.1133. OSHA is proposing to amend section 1910.19 to make it clear that § 1910.1033 is a Subpart Z standard which is incorporated by cross reference into 29 CFR parts 1917 and 1018 for longshoring and marine terminals. OSHA is proposing to amend 29 CFR 1928.21 to indicate that 1910.1033 will be applicable to agriculture. OSHA requests comments on the scope of the proposal and the formal manner by which the standard would be incorporated into the Code of Federal Regulations.

Paragraph (a)(1) proposes to apply all provisions of the standard to "nonindustrial work environments." In addition, paragraph (a)(2) proposes to further extend coverage of the provisions found in paragraph (e)(1), which address exposure to tobacco smoke, to all indoor work environments under OSHA's jurisdiction. This includes indoor work areas on construction sites, shipyards, and agricultural employments. The Agency believes that application of the proposed provisions under paragraph (e)(1) addressing exposure to tobacco smoke is necessary, appropriate, and feasible for any indoor or enclosed workplace covered by OSHA. Compliance with the tobacco smoke provisions essentially entails establishment of a separate enclosure, exhausted directly to the outside, and maintained under negative pressure where smoking is permitted. OSHA sees no feasibility obstacles in application of these provisions to industrial as well as nonindustrial work environments. It is not clear to OSHA, however, that the other indoor air quality provisions of the proposed standard can be feasibly or appropriately applied in typical industrial work environments. These provisions primarily address means to assure effective functioning of HVAC systems and actions felt necessary to be taken to maintain good general indoor air quality. Thus, it may not be feasible or appropriate to apply these provisions to industrial ventilation systems or industrial environments in which control of various industrial contaminant emissions rather than general air quality is the primary issue.

Definitions: Paragraph (b)

The following terms are defined for the purpose of this proposal: "Air Contaminants", "Assistant Secretary", "Building systems", "Building-related illness", "Designated person", "Designated smoking area", "Director", "Employer", "HVAC system", "Nonindustrial work environment", and "Renovation and remodeling".

The term "Air contaminants" refers to substances contained in the vapors from paint, cleaning chemicals, pesticides, solvents, particulates, outdoor air pollutants and other airborne substances which may cause material impairment to the health of employees working within the nonindustrial environment. The term "air contaminants" informs the employer that the provisions addressing control of air contaminants apply to airborne substances which may be within nonindustrial indoor work environments. For purposes of this proposal the definition of air contaminants may be broader than that used in 29 CFR 1910.1000. Hazardous levels of air contaminants may arise from contaminant buildup due to inefficient or insufficient general dilution ventilation with outside air, the misapplication of general dilution ventilation to address strong point sources, indoor activities or operations such as renovation, remodeling, maintenance, etc. which lead to local source emissions, and entry of outdoor contaminants such as vehicle exhausts, wastes, stored materials, or pollutants from adjacent industrial facilities. Provisions are proposed in the standard which require the employer to take measures to address the avenues of contaminant buildup noted above.

The term "Assistant Secretary" means the Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, or designee.

The term "Building systems" applies to the heating, ventilation and air-conditioning (HVAC) system, the potable water system, the energy management system, and all other systems in a facility which may impact indoor air quality. This broad definition was necessary to avoid excluding non-HVAC systems which do impact indoor air quality. In the facilities industry, potable hot water systems are typically considered plumbing systems and not HVAC systems. Plumbing systems (potable hot water) have been implicated in *Legionella* episodes where the water is aerosolized, so excluding plumbing systems from the scope of this standard would have been unacceptable. This definition also intends to focus operation and maintenance efforts on those systems whose failure, degradation, or misuse would adversely impact indoor air quality.

The term "Building-related illness" describes specific medical conditions of known etiology which can be documented by physical signs and laboratory findings. Such illnesses include sensory irritations when caused by known agents, respiratory allergies, nosocomial infections, asthma, humidifier fever, hypersensitivity pneumonitis, Legionnaires' disease, and the symptoms and signs characteristic of exposure to chemical or biologic substances such as carbon monoxide, formaldehyde, chlordane, endotoxins, or mycotoxins. "Building-related illness" defines the medical conditions that, if observed, require evaluation of the facility building systems to determine if they are functioning

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differences, pharmacodynamic processes, and other potential nonlinearities [Ex. 4–57]. PBPK models of nicotine and cotinine have been described for both rats [Exs. 4–112, 4–255[1] and humans [Exs. 4–254, 4–270].

A physiological model of cotinine disposition [Ex. 4–112] was developed to analyze intravenous infusion of nicotine and cotinine and bolus dosing of cotinine in rats. In general, the observed cotinine time profiles in blood and tissues were consistent with linear kinetics, but the distribution of cotinine into all tissues appeared to be roughly three-fold greater following infusion of nicotine than following infusion of cotinine, and the clearance of cotinine following bolus and infusion dosing was significantly different.

A more recent rat model [Ex. 255] featured a physiologically based description of nicotine kinetics and a compartmental description of cotinine. This model provided a successful description of the plasma kinetics of both nicotine and cotinine for intraarterial or intravenous bolus dosing of nicotine. The timecourse of nicotine in most tissues was also consistent with first order kinetics; however, it was necessary to include a description of saturable nicotine binding in the brain, heart, and lung to adequately reproduce nicotine concentration profiles in these tissues. This rat model has also been scaled for use in predicting mouse and human pharmacokinetics [Ex. 4–254]. The human model has recently been expanded to include a physiological description of cotinine as well as a forearm compartment, and is now able to describe nicotine and cotinine kinetics following intravenous infusion of nicotine in humans [Ex. 4–266]. Another human model [Ex. 4–270] has also been developed which includes physiological descriptions of both nicotine and cotinine. This model, which assumes linear kinetics, predicts results which agree with published data on the kinetics of nicotine and cotinine in blood following nicotine infusion as well as cotinine in the blood following the infusion of cotinine.

6. Application of Pharmacokinetic Modeling for ETS Exposure Estimation

Both of the human models described above possess a reasonable biologically based structure, and either model would provide a useful starting point for the development of a PBPK model which could be of use in examining the relationship between cotinine concentrations in body fluids and inhaled nicotine. However, neither of the models currently possesses all of the features which would be necessary for such an analysis. The most useful application of PBPK modeling would appear to be to support an analysis of four issues related to the use of cotinine as a biomarker of ETS exposure: (1) Estimation of the contribution of dietary intake of nicotine to cotinine levels in the plasma, saliva and urine of nonsmokers; (2) Estimation of a plausible upper bound for cotinine concentrations in plasma, saliva and urine associated with ETS exposure (to identify individuals wrongfully identifying themselves as nonsmokers). This can be viewed as a way to validate misclassification results derived from surveys; (3) Evaluation of the potential impact of high affinity, low capacity binding of nicotine and cotinine in nonsmokers with low exposure to ETS; and (4) Evaluation of the potential impact of pharmacokinetic uncertainty and variability on the use of cotinine concentrations in plasma, saliva or urine to infer an individual's ETS exposure. The necessary features for accomplishing these analyses include both inhalation and oral routes of nicotine exposure, a salivary compartment, and a description of nicotine binding in the brain, heart and lung.

In evaluating the use of cotinine as a biomarker of ETS exposure, two kinds of uncertainty must be considered. The first kind of uncertainty embraces those factors which could tend to bias a risk estimate. Two such factors are dietary intake of nicotine and nicotine binding. In both of these cases, the impact of ignoring the effect, if it were significant, would be to overestimate exposure (and therefore risk) for the least exposed individuals. The second kind of uncertainty includes those factors which tend to broaden the confidence interval for the risk estimate. The most significant factors in this category are uncertainty in the fraction of nicotine converted to free cotinine, and the rates of metabolic and urinary clearance of nicotine and cotinine. An example of such uncertainty is results reported for half-lives of cotinine in nonsmokers [Ex. 4–24, 4–73, 4–82, 4–184, 4–186], showing a mean of 16.2 hours, with a coefficient of variation of 0.22.

7. Analysis of Uncertainty

It is useful in this evaluation to distinguish uncertainty from variability. As it relates to the issue of using pharmacokinetic modeling in risk assessment, uncertainty can be defined as the possible error in estimating the "true" value of a parameter for a representative ("average") individual. Variability, on the other hand, represents differences from individual to individual.

For the purpose of evaluating the usefulness of pharmacokinetic modeling for estimating exposure, the uncertainty and variability in the various parameters for the pharmacokinetic models can be grouped into four classes: the physiological parameters (volumes and flows), the tissue distribution parameters (partitioning and binding), and the kinetic parameters (absorption, metabolism, and clearance).

(a) Physiological Parameters. The physiological parameters include (1) the body weight and the weights of the individual organs or tissue groups, (2) the total blood flow and flows to each organ or tissue group, and (3) the alveolar ventilation rate. These quantities have been reasonably well established for the human [Exs. 4–155, 4–309] and the chief effort associated with pharmacokinetic model parameterization in the human is the determination of the necessary level of detail for the physiological description, grouping of the tissues not meriting a separate description into pharmacokinetically similar groups, and the association of the proper volume and flow data with the selected groupings. Existing models for nicotine and cotinine contain a fairly detailed physiological structure and differ only slightly in their assignment of tissues. The model of Plowchalk and deBethizy [Ex. 4–254] includes separate compartments for the brain, heart, and skin. The first two of these tissues are lumped into a "vessel-rich" tissue compartment in the model of Robinson et al. [Ex. 4–270], and the skin is lumped in with the muscle. Conversely, the gastrointestinal tract is given a separate compartment in the Robinson model but is lumped into a "slowly perfused" tissue compartment in the Plowchalk model. These differences mainly reflect the different interests of the modeling groups in terms of target organs and routes of exposure. The Robinson model contains a venous infusion compartment to accommodate the mixing time for arterial administration. The published Plowchalk model does not include this feature, but a forearm compartment has since been added to provide a similar function [Ex. 4–83]. Neither model appears to contain an explicit description of inhalation or oral exposure, but the necessary equations could easily be added to the existing physiological structures. A salivary fluid compartment could also be added to either model if desired. Experience with other chemicals has shown that uncertainty in the physiological

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promulgate standards to assure employees safe and healthful working conditions. Employers must comply with the standards which the Secretary promulgates. The Act defines an employer expansively as a person with employees in a business affecting interstate commerce.

The Agency believes that the proposal as written will protect employees from the risks of poor indoor air quality. Where the owner of a business is not the owner of the space where such business operates, the owner or landlord of the building will probably also be an employer within the meaning of the Act and the definition contained in this proposal. This is so because the building owner or operator will generally have employees (either on site or off site) and will be engaged in a business affecting interstate commerce. In such cases the situation will be construed to be a multi-employer worksite. Such situations are quite common in the context of construction sites. The Agency does not believe that there is any reason to treat nonindustrial multi-employer worksites differently from construction multi-employer worksites for purposes of compliance.

OSHA has a long history of enforcing OSHA standards in multi-employer worksites. Nothing in this proposed rule would change the position that the Agency has taken in cases such as Anning-Johnson (4 OSH Cas. (BNA) 1193, Harvey Workover, Inc., 7 OSH Cas. (BNA) 1687 and in its Field Operations Manual (CPL 2.45 CH-1, Chapter V-9). As a general matter each employer is responsible for the health and safety of his/her own employees. However, under certain circumstances an employer may be cited for endangering the safety or health of another employer's employees. In determining who to hold responsible, OSHA will look at who created the hazard, who controlled the hazard, and whether all reasonable means were taken to deal with the hazard.

It is contemplated that in those cases where there is a multi-employer worksite that the affected employers will divide up the responsibilities in the manner in which they make the most sense. Those who have information at their disposal that is required to be kept under the proposal will make use of the information or make it available to whoever will need that information in the discharge of their duties. For example, the building engineer may have possession of the schematics of the ventilation system. The engineer would make them available to the person responsible for maintaining the system as well as the person responsible for developing the IAQ Compliance Plan (if that is not the same person). The proposal is designed to promote the efficient resolution of indoor air quality problems and will not result in duplicative efforts. There is nothing in the proposal, for example, that would prevent the building owner (who is an employer within the meaning of the Act) from gathering the required information from the various lessee/employers in the premises, developing, and implementing an IAQ Compliance Plan which would be shared with the various employers occupying the premises. In addition, it may be more efficient for the building owner to develop and maintain the records required by the proposal, again sharing them with the various employer-tenants. The Agency believes that the co-operative interrelationships which the performance oriented proposal permits will avoid duplication of compliance activities even within multi-employer worksites.

The term "HVAC system" means the collective components of the heating, ventilation and air-conditioning system including, but not limited to, filters and frames, cooling coil condensate drip pans and drainage piping, outside air dampers and actuators, humidifiers, air distribution ductwork, automatic temperature controls, and cooling towers. This definition also intends to focus on those HVAC system components whose failure, degradation, or misuse would adversely impact indoor air quality.

The term "Nonindustrial work environment" means an indoor or enclosed work space such as, but not limited to, offices, educational facilities, commercial establishments, and healthcare facilities, and office areas, cafeterias, and break rooms located in manufacturing or production facilities. Nonindustrial work environments do not include manufacturing and production facilities, residences, vehicles, and agricultural operations.

The term "Renovation and remodeling" means building modification involving activities that include but are not limited to: removal or replacement of walls, ceilings, floors, carpet, and components such as moldings, cabinets, doors, and windows; painting, decorating, demolition, surface refinishing, and removal or cleaning of ventilation ducts.

The terms "HVAC system", "Nonindustrial work environment", and "Renovation and remodeling" are defined to clarify and illustrate the parameters under which obligations of the standard are incurred. For example, the definition of "HVAC system" lists what OSHA believes to be typical components of such systems which directly affect indoor air quality. These components are enumerated since provisions under the standard propose to require employers to perform routine inspection and maintenance on those components. "Renovation and remodeling" is defined to inform the employer of the situations under which the standard proposes to require the employer to take special precautions when those activities take place.

Indoor Air Quality Compliance Program: Paragraph (c)

This paragraph proposes to require employers to obtain or develop certain written information that will facilitate implementation of measures necessary to prevent degradation of indoor air quality. Paragraph (c)(2) proposes to require the employer to identify a designated person to be given the responsibility of overseeing establishment and implementation of the written compliance program. Paragraph (c)(3) proposes to require the employer to establish a written IAQ compliance program to include at least the following information: a description of the facility building systems; schematics or construction documents locating building systems equipment; information on the daily operation and management of the building systems; a description of the building and its function; a written maintenance program; and a checklist for visual inspection of the building systems. Further, paragraph (c)(4) proposes to require that the following information also must be retained, if available, to assist in indoor air quality evaluations: as built construction documents; HVAC system commissioning reports; HVAC system testing, adjusting and balancing reports; operation and maintenance manuals; water treatment logs; and operator training materials. Paragraph (c)(5) proposes to require the establishment of records of employee complaints of building-related illnesses, as part of the written program.

OSHA believes that written plans are an essential element of an overall compliance program since it will encourage employers to focus on indoor air quality and implement the necessary controls and measures to achieve compliance with the standard [Exs. 3-38, 3-85, 3-412, 3-434, 3-500, 3-502, 3-505, 3-529]. The development of documented safety and health programs and procedures is a well-established and common practice in industry, and requirements for written programs are typically found in other OSHA standards dealing with exposure to toxic

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hazardous substance can only file a product liability suit against a third party manufacturer, processor, distributor, sales firm, or contractor. It is often difficult, however, to demonstrate a direct link between an exposure to a hazardous substance and the illness.

In order to pursue litigation successfully, there must be specific knowledge of the magnitude and duration of a worker's exposure to a hazardous substance, as well as the causal link between the disease and the occupational exposure. Usually, it is extremely difficult to isolate the role of occupational exposures in causing the disease, especially if workers are exposed to many toxic substances and the exposure is not necessarily limited to the workplace such as the case for ETS. This difficulty is further compounded by the long latency periods that are frequently involved. In addition, the liable party must be identifiable, but workers may have several employers over a working lifetime. The burden of proof that an occupational exposure to a hazardous substance occurred, that a specific employer is the liable party, and that the exposure level was significant may prohibit the individual from initiating the suit.

There are an increasing number of lawsuits that are related to health effects to building occupants from poor indoor air quality. These lawsuits are typically filed after the illness or health effect has been diagnosed. In this sense, increasing pressure is being placed on businesses. However, the legal pressure currently does not relate to the implementation of a clean indoor air policy (e.g., legal action is not currently being taken just because a company does not have a clean indoor air policy. These actions are event related as opposed to being policy related). IAQ litigation is growing rapidly and the focus is shifting from residential to commercial facilities. Examples to emphasize that are the recent $12.5 million claims against the Social Security Administration for the Richmond, California episode of Legionnaire's disease, the Call versus Prudential case in which building tenants settled with the defendants in what may have been the first jury trial in sick building litigation, and a suit by Hamilton, Ohio, county employees against their office building owners alleging exposure to fumes, bacteria, fungi, dust and irritants [Ex. 3–575].

Legal proceedings do not internalize occupational illness costs because they involve substantial legal fees associated with bringing about court action. In deciding whether to sue, the tort victim must be sure that the size of the claim will be large enough to cover legal expenses. In effect, the plaintiff is likely to face substantial transaction costs in the form of a contingency fee, commonly 33 percent, plus additional legal expenses. The accused firm must also pay for its defense. The high costs and uncertainties associated with tort law make it an inefficient mechanism for ensuring adequate protection of workers' health.

Insurance and liability costs are not borne in full by the specific employer responsible for the risk involved. For firms that are insured, the premium determination process is such that premiums only partially reflect changes in risk associated with changes in exposure to hazardous substances. This lack of complete adjustment is the so-called "moral hazard" problem, which is the risk that arises from the possible imprudence of the insured. As the insured firm has paid an insurance company to assume some of the risks, that firm has less reason to exercise the diligence necessary to avoid losses. Transfer of risk is a fundamental source of imperfection in markets.

There is a growing number of state and local laws and ordinances controlling smoking. Armed with new data that show health effects from indoor air pollutants, plaintiffs who believe that they have been injured by the air inside their workplaces are beginning to take the offensive. They are lobbying on the local, state and federal levels for protective legislation, and in the absence of such legislation, they are suing for damages to their health. These cases are complex not only in the nature of the technical proof that must be developed and presented, but also in the number of parties involved. Suits have been filed against architects, builders, contractors, building product manufacturers and realtors [Ex. 3–662].

(b) Workers' Compensation. The Workers' Compensation system is a result of the perceived inadequacies in liability or insurance systems to compel employers to prevent occupational disease or compensate workers fully for their losses. The system was designed to internalize some of the social costs of production, but in reality it has fallen short of compensating workers adequately for occupationally related disease. Thus, society shares the burden of occupationally related health effects, premature mortality, excess morbidity, and disability through taxpayer support of social programs such as welfare, Social Security disability payments, and Medicare.

Compensation tends to be inadequate especially in permanent disability cases, in view of the expiration of benefit entitlement and the failure to adjust benefits for changes in a worker's expected earnings over time. As of January 1987, eight states restricted permanent disability benefits either by specifying a maximum number of weeks for which benefits could be paid or by imposing a ceiling on dollar payments [Ex. 4–302].

At present, time and dollar restrictions on benefit payments are even more prevalent in the area of survivor benefits. The duration of survivor benefits is often restricted to 10 years, and dollar maximums on survivor payments range from $7,000 to $60,000. In addition, it should be noted that if the employee dies quickly from the occupational illness and has no dependents, the employer need pay only nominal damages under Workers' Compensation (e.g., a $1,000 death benefit).

Finally, in spite of current statutory protection, disability from occupational diseases represents a continuing, complex problem for Workers' Compensation programs. Occupational diseases may take years to develop, and more than one causal agent may be involved in their onset. Consequently, disabilities resulting from occupationally induced illness often are less clearly defined than those from occupationally induced injury. As a result, Workers' Compensation is often a weak remedy in the case of occupational disease. Indeed, there is some evidence indicating that the great majority of occupationally induced illnesses are never reported or compensated [Ex. 4–84].

The insurance premiums paid by a firm under the Workers' Compensation system are generally not experience rated; that is, they do not reflect the individual firm's job safety and health record. About 80 percent of all firms are ineligible for experience rating because of their small size. Such firms are class rated, and rate reductions are granted only if the experience of the entire class improves. Even when firms have an experience rating, the premiums paid may not accurately reflect the true economic losses. Segregation of loss experience into classes is somewhat arbitrary, and an individual firm may be classified with other firms that have substantially different normal accident rates. An experience rating is generally based on the benefits paid to workers, not on the firm's safety record. Thus, employers may have a greater incentive to reduce premiums by contesting claims than by initiating safety measures.

In summary, the Workers' Compensation system suffers from

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TABLE VI–10.—COST FOR SYSTEM OPERATION AND MAINTENANCE PROVISION—Continued

	Buildings with IAQ problems and without HVAC maintenance			Buildings without HVAC maintenance and without IAQ problems		Total annual cost ($ million)
	No. of buildings with HVAC	Annualized[1] cost to improve IAQ ($ million)	Annual[2] cost ($ million)	No. of buildings	Annual cost[3] ($ million)	
Government	87,086	163	256	203,200	597	1,017
Total	683,404	1,281	2,009	1,594,610	4,688	7,979

[1] Number of problem buildings x $1.14 per sq. ft. x 14,000 sq. ft. (mean floorspace/building), annualized over 20 years at 10% interest rate.
[2] Recurring cost estimated for number of problem buildings without HVAC maintenance using $0.21/sq. ft.
[3] Number of non-problem buildings without maintenance x 14,000 sq. ft. (mean floorspace per building) x $0.21 per square foot.
Source: OSHA, Office of Regulatory Analysis, 1994.

OSHA assumed that the labor costs for the trainer and preparation time are approximately equal to 25 percent of the trainee's wage cost during the session. OSHA also assumed that 50 percent of the workers will require such training. The cost equation for maintenance workers training is as follows:

$C_t = N_m \times P_m \times W_m \times T_m$

where

C_t=the cost of training of maintenance workers
N_m=the number of maintenance workers[10]
P_m=the percentage of existing compliance as estimated by OSHA (50 percent)
W_m=the hourly compensation wage rate for maintenance workers ($10.95)
T_m=one-half hour of maintenance worker time plus 7.5 minutes for trainer cost (37.5 minutes)

The total cost of training is estimated at $6.84 per maintenance worker for a half hour program. Table VI–11, presents OSHA's estimate for the number of maintenance workers needing training and the associated costs. The annualized first year cost is estimated at $0.5 million. It was assumed that job changes within establishments or buildings will require retraining. The annual new hire cost is estimated at $0.8 million, based upon industry turnover rates. Thus OSHA estimated annualized cost training to be $1.3 million.

The proposed standard requires employers to inform all employees of the contents of the standard and its appendices. This could be accomplished by posting the proposed standard at a bulletin board; therefore, OSHA did not include a cost for this provision.

(d) Compliance with Related Standards. The proposed standard requires employees performing work on HVAC systems to comply with several existing OSHA standards and therefore any costs associated with compliance with this provision have already been considered. This requirement is necessary to protect employees from exposure to indoor air pollutants and exposure to noise. This provision is considered to have a de minimus effect on all industries and OSHA believes that establishments are in full compliance with this requirement.

(e) Air Contaminant-Environmental Tobacco Smoke. The primary objective of the tobacco smoke provision is to eliminate the nonsmoker's exposure to ETS. Under the proposed rule, firms will have the option of either banning smoking of tobacco products or permitting smoking only in designated areas.

OSHA recognizes that not all establishments will make available designated smoking areas as there may be physical constraints on the option of providing separate ventilation. Such constraints are imposed by the building's design, the building's mechanical ventilation system's capabilities, by costs involved in providing adequate ventilation, by the occupant use of the building. In some cases, establishments located in severe climate zones may find it necessary to protect their smoking employees from weather exposure by providing designated smoking areas.

TABLE VI–11.—TRAINING COST FOR MAINTENANCE WORKERS

	Building maintenance workers	Maintenance employees to be trained[1]	Annualized initial cost[2] ($ million)	Annual new hire training cost ($ million)	Annual cost ($ million)
Agriculture, forestry, fishing	26,210	13,105	$0.015	$0.01	$0.01
Mining	5,460	2,730	0.003	0.00	0.01
Construction	73,060	36,530	0.041	0.07	0.11
Manufacturing	205,660	102,830	0.115	0.18	0.29
Transportation	47,720	23,860	0.027	0.02	0.04
Wholesale and retail trade	143,440	71,720	0.080	0.12	0.20
Finance, insurance, real estate	172,350	86,175	0.096	0.17	0.26
Services	236,160	118,080	0.132	0.26	0.39
Government	NA	NA	NA	NA	NA
Total	910,060	455,030	0.507	0.82	1.31

[1] Based on preliminary OSHA estimate of 50 percent existing compliance.

[10] The number of maintenance workers is based on BLS's Occupational Employment Statistics survey of 1992 and includes all maintenance workers who perform work involving two or more maintenance skills to keep machines, mechanical equipment, or structure of an establishment in repair.

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the functioning of the HVAC system and determine if the employees are affected.

APPENDIX B to §_____._____—INFORMATION SOURCES—NONMANDATORY

The following is a partial list of available data sources which building owners/agents of employers may wish to consult to help identify, characterize, and reduce sources of indoor air pollutants in office work environments. These sources also provide useful information concerning the operation, maintenance, and evaluation of mechanical ventilation systems.

Building Air Quality: A Guide for Building Owners and Facility Managers. U.S. EPA/NIOSH. Dec. 1991. EPA/400/1–91/033.. DHHS (NIOSH) Publication No. 91–114. Available from Superintendent of Documents, P.O. Box 371954, Pittsburgh, PA 15250–7954.

Introduction to Indoor Air Quality: (1) Self-Paced Learning Module and (2) A Reference Manual. U.S. EPA, Office of Air and Radiation. EPA/400/3–91/00. July 1991.

Managing Indoor Air Quality. 1991. Shirley J. Hansen. The Fairmont Press, Inc., 700 Indian Trail, Lilburn, GA 30247.

ASHRAE Standard 62–1989. Ventilation for Acceptable Indoor Air Quality. American Society of Heating, Refrigeration, and Air-conditioning Engineers, Inc. 1791 Tullie Circle, NE, Atlanta, GA 30329.

Washington State Ventilation and Indoor Air Quality Code, Chapter 51–13 WAC. Washington State Building Code Council.

Indoor Air Quality Workbook. 1990. D. Jeff Burton. IVE, Inc., 178 North Alta Street, Salt Lake City, Utah 84103.

APPENDIX C to §_____._____—SMOKING CESSATION PROGRAM INFORMATION—NONMANDATORY

The following organizations provide smoking cessation information and program material:

(1) The National Cancer Institute operates a toll-free Cancer Information Service (CIS) with trained personnel to help you. Call 1–800–4–CANCER to reach the CIS office serving your area, or write: Office of Cancer Communications, National Cancer Institute, National Institutes of Health, Building 31, Room 10A24, Bethesda, Maryland 20892.

(2) American Cancer Society, 1599 Clifton Road NE, Atlanta, Georgia 30062, (404) 320–3333. The American Cancer Society (ACS) is a voluntary organization composed of 58 divisions and 3,100 local units. Through "The Great American Smokeout" in November, the annual Cancer Crusade in April, and numerous educational material, ACS helps people learn about the health hazards of smoking and become successful exsmokers.

(3) American Heart Association, 7320 Greenville Avenue, Dallas Texas 75231, (214) 750–5300. The American Heart Association (AHA) is a voluntary organization with 130,000 members (physicians, scientists, and laypersons) in 55 states and regional materials about the effects of smoking on the heart. AHA also has developed a guidebook for incorporating a weight-control component into smoking cessation programs.

(4) American Lung Association, 1740 Broadway, New York, New York 10019, (212) 245–8000. A voluntary organization of 7,500 members (physicians, nurses and laypersons), the American Lung Association (ALA) conducts numerous public information programs about the health effects of smoking. ALA has 59 state and 85 local units. The organization actively supports legislation and information campaigns for nonsmokers' rights and provides help for smokers who want to quit, for example through "Freedom From Smoking," a self-help cessation program.

(5) Office on Smoking and Health, United States Department of Health and Human Services, 5600 Fisher Lane, Park Building, Room 110, Rockville, Maryland 20857. The Office of Smoking and Health (OSH) is the Department of Health and Human Services' lead agency in smoking control. OSH has sponsored distribution of publications on smoking-related topics, such as free flyers on relapse after initial quitting, helping a friend or family member quit smoking, the health hazards of smoking, and the effects of parental smoking on teenagers.

PART 1928—OCCUPATIONAL SAFETY STANDARDS FOR AGRICULTURE—AMENDED

7. The authority citation for Part 1928 is proposed to continue to read as follows:

Authority: Secs. 4, 6, 8, Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657); Secretary of Labor's order Nos. 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736), or 1–90 (55 FR 9033), as applicable; 29 CFR part 1911.

8. Section 1928.21 is proposed to be amended by adding a new paragraph (a)(6) as follows:

§ 1928.21 Applicable standards in 29 CFR Part 1910.

(a) ***

(6) Indoor air quality—Section 1910.1033.

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hours. This strategy, however, applies only to occupant generated contaminants like carbon dioxide and odors. Housekeeping cleaning agents or pesticides are typical of contaminants that may be released which could not be absorbed by a non-ventilated space. Consequently, other contaminants must be diluted/removed by the ventilation system whenever the building is occupied. In addition, it is recognized that certain automatic temperature control strategies can also prevent a facility from receiving the minimum outside air ventilation rate. The obvious example is the early morning warm-up cycle wherein the outside air dampers are kept shut in the morning until the space temperature recovers from the setback temperature of the night before. These energy conservation and temperature control strategies must not interfere with providing minimum outside air ventilation when the building is occupied.

Paragraph (d)(4) proposes to require the employer to utilize general or local exhaust ventilation, as provided by the existing HVAC system or auxiliary systems, to minimize the hazards associated with maintenance or housekeeping activities. The literature reports IAQ/BRI episodes that were initiated with activities like painting, carpet cleaning and floor resurfacing. If these activities occur during unoccupied periods then chemical vapors from paints and adhesives and excessive moisture from carpet cleaning may be diluted and removed by the outside air ventilation function of the HVAC system. During occupied periods, efforts should be made to restrict transportation of hazardous contaminants from these activities throughout the facility by the HVAC air distribution system.

Paragraph (d)(5) proposes to require the employer to maintain occupied space relative humidities below 60% in buildings with mechanical cooling systems. Moisture in a building may support and amplify microbial contamination with potential for aerosolization. Both the American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc. (ASHRAE) in their Standard 62–1989 titled "Ventilation for Acceptable Indoor Air Quality", section 5.11 [Ex. 4–333] and the American Conference of Governmental Industrial Hygienists (ACGIH) in their 1989 "Guidelines for the Assessment of Bioaerosols in the Indoor Environment" [Ex. 3–61] recommend that relative humidity in the occupied space be maintained below 60%.

OSHA is inviting comments on whether a relative humidity of 60% is the appropriate upper limit to inhibit microbial growth or if a higher limit is appropriate. In addition, OSHA would like comment on whether there should be a lower level of relative humidity as recommended by ASHRAE and ACGIH to reduce irritation effects due to low relative humidity. And finally, OSHA would like additional comment on whether it is feasible in hot and humid climates to achieve relative humidities of 60% or less.

Paragraph (d)(6) proposes to require the employer to monitor for carbon dioxide (CO_2) in the occupied space as part of maintenance or employee complaint investigations. When the concentration exceeds 800 ppm, the employer would be required to check the operation of the HVAC system. CO_2 is frequently used as a gross surrogate indicator of indoor air quality. Ideally, by knowing the rate of accumulation of CO_2 in the space and the rate of generation of CO_2 by respiring occupants in the space, it would be possible to predict the rate of removal of CO_2 from the space by the HVAC system. Because buildings have average occupant densities to generate CO_2, the concentration is an indicator of the HVAC system's ability to dilute and remove occupant generated contaminants like CO_2, water vapor, and odors (human bioeffluents). However, the CO_2 concentration and the associated outside air ventilation rate offers no confidence as to the adequacy of dilution and removal of other contaminants released in the space. If the outside air ventilation rate is insufficient to dilute and remove CO_2, then it can be assumed that other contaminant concentrations will also be elevated. The literature reports that CO_2 concentrations in the space under 800 ppm will minimize health-related complaints [Exs. 3–34A, 4–331].

Paragraph (d)(8) proposes to require the employer to restrict the presence of hazardous substances in air distribution systems. The HVAC air distribution system itself should not be the source of hazardous contaminants due to its' critical nature as a potential pathway to building occupants. Enclosed ducts are typically not used to store hazardous substances but non-ducted air transport pathways such as area-ways, plenums, chases, corridors, and mechanical rooms serving as return air plenums are sometimes used for storage. If these air transport pathways are used for storage, then the employer must be especially careful to make sure that no spillage or leakage of hazardous substances occurs. This will insure that the pathways are kept free of hazardous substances.

Paragraph (d)(11) proposes to require that employees working on building systems are provided with and use personal protective equipment (PPE) as required by other OSHA standards including; 29 CFR 1926, Subpart E, Personal Protective and Life Saving Equipment; 29 CFR 1926.52, Occupational Noise Exposure; 29 CFR 1910, Subpart I, Personal Protective Equipment; and 29 CFR 1910.95 Occupational Noise Exposure.

OSHA is aware, through its experience and through the literature and submissions to the docket, that HVAC Operations and Maintenance (O&M) personnel may often receive minimal training regarding existing relevant OSHA regulations and the hazards that they are exposed to in the performance of their duties. Sometimes, facilities are not viewed as industrial workplaces by either the management or employees. However, the hazards do exist and therefore compliance with existing regulations is necessary to protect the health and safety of O&M employees. Respirators may not normally be used in this industry due to the perceived lack of a substance-specific hazard. But situations may occur, for instance, such as chemical or microbial contamination, that would require compliance with 1910.134.

Other provisions of this section require; that buildings without mechanical ventilation be operated and maintained to provide natural ventilation; that inspections and maintenance of building systems be performed by or under the supervision of the designated person; that the employer establish a written record of building system inspections and maintenance required under this section; that the employer evaluate the need to perform modifications to the building systems to meet the minimum requirements specified in paragraph (d) of this section in response to employee complaints of building-related illnesses.

Controls for Specific Contaminant Sources: Paragraph (e)

This paragraph proposes to require employers to take specific protective measures to control employee exposure to specific agents such as tobacco smoke [Exs. 3–7, 3–10, 3–85, 3–291, 3–305, 3–409, 3–449, 3–496, 3–505B], outdoor pollutants [Ex. 3–496, 3–500, 3–502, 3–505], contaminant emissions from local indoor sources [Exs. 3–10, 3–17, 3–26, 3–38, 3–412], microbial contaminants [Exs. 3–10, 3–26, 3–61, 3–496, 3–500, 3–502, 3–505, 3–506], hazardous chemicals including cleaning and

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parameters generally has much less impact on overall model uncertainty because they are known relatively well and are not as influential on model behavior as the distribution and kinetic parameters [Ex. 4–56].

(b) Distributional Parameters. In both of the published human models, the tissue partitioning was initially estimated on the basis of steady-state tissue/blood concentration ratios measured in animals. The partitioning parameters in the Robinson model were then iteratively adjusted to fit other timecourse data. The resulting partition coefficients in the two models differ by a factor from two to five in corresponding tissues. The partitioning data for cotinine, determined by Gabrelsson and Bondesson [Ex. 4–112], show a similar level of uncertainty; partitions for cotinine following infusion of nicotine were two- to five-fold higher than the same partitions following infusion of cotinine. The lack of reproducibility of these data represents a deficiency in the development of PBPK modeling for these chemicals. Fortunately, the partition coefficients tend to be less important than the kinetic parameters in terms of overall model performance. To a large extent, as long as the volume of distribution associated with the physiological structure and partition coefficients is in agreement with the apparent pharmacokinetic volume of distribution for each chemical, the model will perform adequately in terms of timecourses in blood and urine. This was evidenced by the ability of the Robinson model to reproduce published nicotine and cotinine pharmacokinetic data [Ex. 4–270]. A potentially more significant uncertainty associated with distribution is the possibility of pharmacokinetically significant tissue binding of nicotine. Satisfactory description of the timecourse of nicotine in the brain, lung, and heart of the rat required the inclusion of binding in these tissues [Ex. 4–255]. Clearly, the relatively low capacity, high affinity binding associated with nicotine is unlikely to effect total systemic clearance except at very low concentrations. However, the existence of nonlinear pharmacokinetics at low concentrations could lead to a miscalculation of exposure for the least exposed individuals. It has been suggested that there is a longer clearance half-life for nicotine, and therefore cotinine, associated with low circulating concentrations, and that this longer half-life is due to the slower release of nicotine bound to tissues [Exs. 4–28, 4–24, 4–167]. To date, no careful pharmacokinetic investigation of this possibility has been performed in the human model, and adequate nicotine-specific tissue binding information does not appear to have been collected except perhaps in the brain.

(c) Kinetic Parameters. By far the most significant parameters in the models are those describing the absorption, metabolism, and clearance of nicotine and cotinine. The Robinson model uses reported human hepatic and renal clearance values for nicotine and cotinine. The sensitivity of this model to these input parameters was investigated by varying them within the range of reported clearance values from infusion studies in humans. The resulting model predictions for post-infusion blood levels, urinary output, and the elimination half-lives of both nicotine and cotinine were found to be well within the ranges of those observed in human studies. Thus the model structure does not produce an exaggerated response to variation of the input parameters, and reflects the natural interaction between measures of clearance, volume of distribution, and rates of elimination. In the case of the physiological parameters, variability dominates over uncertainty, while for the distributional parameters, uncertainty dominates. In the case of the kinetic parameters describing clearance, it appears that variability again dominates. For example, the mean values for the terminal half-life of cotinine reported in different studies range from 12 to 21 hours in non-smokers [Exs. 4–24, 4–73, 4–82, 4–184, 4–186]. The coefficient of variation in these same studies, a measure of interindividual variability, ranges from 17–22%, and the coefficient of variation for the entire collection of reported individual values is similar: 22% (N=35, mean=16.2). A review of the published data on infusion of nicotine and cotinine in humans [Ex. 4–270] found a 3-fold variation in reported half-lives for cotinine. For comparison, the variation in the volume of distribution for cotinine was 5-fold, while for the half-life and volume of distribution of nicotine, the variation was 8-fold and 6-fold, respectively. An even greater level of variability can be expected for the kinetic parameters for the renal clearance of nicotine and cotinine.

OSHA considers the use of pharmacokinetics and specifically PBPK models an important tool in characterizing and quantifying internal dose for evaluation potential exposures and seeks comment on the applicability of this approach in ascertaining the relationship between adverse health effects and exposure to ETS.

V. Significance of Risk

Before the Secretary can promulgate any permanent health or safety standard, he must find that a significant risk of harm is present in the workplace and that the new standard is reasonably necessary to reduce or eliminate that risk. *Industrial Union Department, AFL-CIO* v. *American Petroleum Institute,* 444 U. S. 607, 639–642 (1980) (Benzene). In the Benzene case, the Supreme Court held that section 3(8) of the Act, which defines a "occupational safety and health standard" as a "requirement reasonably necessary or appropriate" to promote safety or health requires that, before promulgating a standard, the Secretary must find, "on the basis of substantial evidence, that it is at least more likely than not that long-term exposure to [the hazard without new regulation] presents a significant risk of material health impairment." 444 U. S. at 653.

In the Benzene decision, the Supreme Court indicated when a reasonable person might consider the risk significant and take steps to decrease it. The Court stated:

> It is the Agency's responsibility to determine in the first instance what it considers to be a "significant" risk. Some risks are plainly acceptable and others are plainly unacceptable. If, for example, the odds are one in a billion that a person will die from cancer by taking a drink of chlorinated water, the risk clearly could not be considered significant. On the other hand, if the odds are one in a thousand that regular inhalation of gasoline vapors that are 2% benzene will be fatal, a reasonable person might well consider the risk significant and take the appropriate steps to decrease or eliminate it. (*IUD* v. *API,* 448 U. S. at 655).

A. Environmental Tobacco Smoke

Two of the adverse health effects associated with exposure to ETS are lung cancer and heart disease (coronary heart disease, excluding strokes). Clinically, lung cancer is almost always fatal. However, heart disease runs the gamut from severe to disabling to fatal. Both of these diseases then constitute the type of "material impairment of health or functional capacity" which the Act seeks to reduce or eliminate. Therefore a standard aimed at reducing the incidence of these impairments is an appropriate exercise of the Secretary's regulatory authority.

In the case before us the Agency estimates that there will be approximately between 144 and 722 cases of lung cancer per year among nonsmoking American workers exposed to ETS in the workplace. When considered over a working lifetime, this translates into an excess lung cancer

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Such an exposure can be measured by the determination of the absorption, distribution, metabolism and excretion of tobacco smoke constituents and/or their metabolites. However, relatively few of these individual constituents have been identified and characterized. Also, measurement of all components in tobacco smoke is not feasible. Therefore, it becomes necessary to identify a marker which, when measured, will accurately represent the frequency, duration and magnitude of the exposure to environmental tobacco smoke.

This discussion reviews available data for the purposes of assessing exposure to ETS in the workplace. Nonsmokers are exposed to mainstream smoke after it has been exhaled by smokers, and to diluted sidestream smoke. Issues covered include activity patterns affecting the duration of nonsmokers' exposures, the concentrations of ETS in buildings, the comparison of ETS components in indoor workplaces, levels of biomarkers in workers, and the inadequacy of general dilution ventilation to address ETS exposure control. This discussion will indicate not only that exposure occurs, but that nonsmokers absorb ETS components.

(a) Chemistry. Pipe, cigar, and cigarette smoke all contribute to environmental tobacco smoke (ETS) but cigarette smoke is of principal interest because it is by far the most common. Tables III–6 and III–7 list some of the known constituents of tobacco smoke.

The combustion of tobacco leads to the formation of mainstream smoke (MS) and sidestream smoke (SS). MS is generated during puff-drawing in the burning cone and hot zones; it travels through the tobacco column and is inhaled by the smoker. The smoke which is exhaled by the smoker, while different from the inhaled smoke, is also considered "mainstream." SS is formed in between puff-drawing and is emitted directly from the smoldering tobacco product into the ambient air.

TABLE III–6.—VAPOR PHASE CONSTITUENTS OF TOBACCO SMOKE AND RELATED HEALTH EFFECTS

Constituent	Amount in MS	Ratio in SS/MS	Health effects
Carbon monoxide	10–23 mg	2.5–4.7	Nervous system, cardiovascular system.[1]
Carbon dioxide	20–40 mg	8–11	Nervous system, cardiovascular system.[1]
Carbonyl sulfide	12–42 µg	0.03–0.13	Irritant, cardiovascular, and nervous systems.[1]
Benzene	12–48 µg	5–10	Known human[3] carcinogen.
Toluene	100–200 µg	5.6–8.3	Irritant, nervous system.[1]
Formaldehyde	70–100 µg	0.1–50	Probable human carcinogen.[3]
Acrolein	60–100 µg	8–15	Irritant, pulmonary.[1]
Acetone	100–250 µg	2–5	Irritant.[1]
Pyridine	16–40 µg	6.5–20	Irritant, nervous system, liver, kidney.[1]
3-methylpyridine	12–36 µg	3–13	Irritant.[2]
3-vinylpyridine	11–30 µg	20–40	Irritant.[2]
Hydrogen cyanide	400–500 µg	0.1–0.25	Irritant, nervous, cardiovascular and pulmonary system.[1]
Hydrazine	32 ng	3	Probable human carcinogen.[3]
Ammonia	50–130 µg	3.7–5.1	Irritant.[1]
Methylamine	11.5–28.7 µg	4.2–6.4	Irritant.[1]
Dimethylamine	7.8–10 µg	3.7–5.1	Irritant[1].
Nitrogen oxides	100–600 µg	4–10	Pulmonary and cardiovascular system.[1]
N-nitrosodimenthylamine	10–40 ng	20–100	Probable human carcinogen.[3]
N-nitrodiethylamine	ND–25 ng	<40	Probable human carcinogen.[3]
N-nitrosopyrrolidine	6–30 ng	6–30	Probable human carcinogen.[3]
Formic acid	210–490 µg	1.4–1.6	Irritant, skin, kidney, liver[1].
Acetic acid	330–810 µg	1.9–3.6	Irritant.[1]
Methyl chloride	150–600 µg	1.7–3.3	Nervous system.[1]
1,3-butadiene	69.2 µg	3–6	Probable human carcinogen.[3]

[1] NIOSH Pocket Guide to Chemical Hazards. U.S. Department of Health and Human Services. Public Health Services, 1990. Ex. 4–238.
[2] Hazards in the Chemical Laboratory. Ed: L. Bretherick, The Royal Society of Chemistry, 1986. [Ex. 4–137]
[3] EPA: Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders, 1992. [Ex. 4–311]

TABLE III–7.—PARTICULATE PHASE CONSTITUENTS OF TOBACCO SMOKE AND RELATED HEALTH EFFECTS

Constituent	Amount in MS	Ratio in SS/MS	Health effects
Particulate matter contains di- and polycyclic aromatic hydrocarbon.	15–40 mg	1.3–1.9	Animal carcinogen.[4]
Nicotine	1–2.5 mg	2.6–3.3	Nervous and cardiovascular system.[1]
Anatabine	2–20 µg	<0.01–0.5	N/A.[5]
Phenol	60–140 µg	1.6–3.0	Irritant.[1]
Catechol	100–360 µg	0.6–0.9	Irritant.[3]
Hydroquinone	110–300 µg	0.7–0.9	N/A.[5]
Aniline	360 ng	30	Probable human carcinogen.[4]
2-Toluidine	160 ng	19	Irritant, cardiovascular system.[1]
2-Naphthylamine	1.7 ng	30	Known human carcinogen.[4]
4-Aminobiphenyl	4.6	31	Known human carcinogen.[4]
Benz[a]anthracene	20–70 ng	2–4	Animal carcinogen.[4]
Benzo[a]pyrene	20–40 ng	2.5–3.5	Probable human carcinogen.[4]
Cholesterol	22 µg	0.9	N/A.[5]
Γ-butyrolactone	10–22 µg	3.6–5.0	Animal carcinogen.[4]
Quinoline	0.5–2 µg	3–11	Irritant.[3]
Harman [1-methyl-9H-pyrido[3,4-b]-indole	1.7–3.1 µg	0.7–1.7	N/A.[5]
N-nitrosonornicotine	200–3000 ng	0.5–3	Animal carcinogen.[4]

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Air Quality During Renovation and Remodeling: Paragraph (f)

Paragraph (f)(1) proposes to require implementation of specific procedures to minimize degradation of air quality during renovation and remodeling activities (Exs. 3–26, 3–38, 3–444B).

Paragraph (f)(2) proposes to require development and implementation of a work plan to restrict entry of air contaminants into other work areas during remodeling, renovation, and similar activities (Ex. 3–444b). Where appropriate, elements of the workplan to be considered are requirements of this standard, implementation of means to assure that HVAC systems continue to function effectively during remodeling and renovation activities, isolation or containment of work areas and appropriate negative pressure containment, air contaminant suppression controls or auxiliary air filtration, and controls to prevent air contaminant entry into HVAC systems. Finally, paragraph (e)(3), proposes to require 24 hour advance notification of employees, or promptly in emergency situations, of work to be performed on the building that may introduce air contaminants into their work area. Such notification must include anticipated adverse impacts on indoor air quality or workplace conditions.

The provisions under proposed paragraphs (f)(1) and (f)(2) are intended to ensure that renovation, remodeling and similar activities are performed in a manner that will reduce the potential for air contaminants generated during those activities from entering other areas of the building. Such activities which may involve demolition, sanding, surface refinishing, component removal and replacement, etc. can result in hazardous substance emission from solvents, paints, carpets, etc. and can also produce high levels of particulate contamination. To control such emissions, the standard proposes to require employers to develop a workplan for the implementation of appropriate work procedures and controls such as exhaust ventilation, isolation, containment, or use of wet methods during renovation and remodeling activities.

Finally, paragraph (f)(3) proposes to require notification of employees in the vicinity of renovation and remodeling activities who may be subject to incidental exposure to emissions produced during such activities (Ex. 3–444B). This notification must also apprise affected employees of the potential adverse impact on air quality. Informing employees of potential workplace hazards is felt by the Agency to be imperative for the success of any safety and health program. OSHA believes that employees can do much to protect themselves if they are informed of the nature of the hazards to which they are exposed.

Employee Information and Training: Paragraph (g)

Paragraph (g) proposes to require employers to provide special training for workers involved in maintenance activities and those involved in HVAC system operations, and to provide certain pertinent information to all employees.

Paragraph (g)(1) proposes to require that maintenance and HVAC operations personnel be trained in the use of personal protective equipment (PPE) required to be worn; training on how to maintain adequate ventilation of exhaust fumes during building cleaning and maintenance; and training of maintenance personnel on how to minimize adverse effects on indoor air quality during the use and disposal of chemicals and other agents [Exs. 3–26, 3–38, 3–41, 3–347, 3–415, 3–434, 3–440, 3–444B, 3–500, 3–502].

Paragraph (g)(2) proposes to require that all employees shall be informed of the contents of the standard and its appendices, signs and symptoms associated with building-related illness, and the requirement under proposed subparagraphs (d)(12) and (d)(13) which directs the employer to evaluate the effectiveness of the building systems, if necessary, upon receipt of complaints from employees of building-related illness [Exs. 3–38, 3–347, 3–412, 3–415, 3–434, 3–444B, 3–500, 3–529]. The information proposed to be provided under this subparagraph need not be conveyed to employees through formal training sessions or courses. Informing employees can be accomplished, for example, through written means such as fact sheets, memos, or posted bulletins. OSHA will provide in a non-mandatory appendix to the final rule an example illustrating what information is to be provided to employees.

Paragraph (g)(3) proposes to require that the employer make training materials developed under these provisions, including the standard and its appendices, available for inspection and copying by employees, designated employee representatives, the Director, and the Assistant Secretary.

Training and information requirements are routine components of OSHA health standards. The inclusion of training and information requirements reflects the Agency's conviction, as noted above, that informed employees are essential to the operation of any effective health program. OSHA believes that informing and training employees about the hazards to which they are exposed will contribute substantially to reducing the incidence of diseases caused by workplace conditions. Further, as noted earlier, it has been OSHA's experience that unacceptable indoor air quality is often the result of deficiencies in implementing effective HVAC system operation and maintenance programs. The Agency believes that specialized training of workers performing those activities is, therefore, necessary to ensure successful performance of their jobs.

Recordkeeping: Paragraph (h)

Paragraph (h) proposes to require that employers maintain records of: All written information regarding the IAQ compliance program required to be established under paragraph (c); inspection and maintenance records required to be established under paragraph (d) [Ex. 3–26], which must include the specific remedial or maintenance actions taken, the name and affiliation of the individual performing the work, and the date of the inspection or maintenance activity; and records of employee complaints of building-related illness required to be established under paragraph (c)(5) of this section [Ex. 3–502].

Paragraph (h) also proposes to require the employer to retain these for at least the previous three years [Ex. 3–502], except that operation, maintenance, inspection, and compliance program records need not be retained for three years if rendered obsolete by the establishment and replacement of more recent records, or rendered irrelevant due to HVAC system replacement or redesign. The records required to be maintained by the employer are to be made available to employees and their designated representative and the Assistant Secretary for examination and copying.

Finally, paragraph (h)(6) proposes to require that whenever the employer ceases to do business records that are required to be maintained by the employer are to be provided to and retained by the successor employer [Ex. 3–440B].

Section 8 (c) of the Act authorizes OSHA to require employers to make, keep, and preserve, and make available to the Secretary or the Director records regarding their activities as prescribed by regulation as appropriate and necessary for the enforcement of the Act or for developing information regarding the causes and prevention of occupational illnesses. As noted earlier,

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rate in the workplace of one per thousand. As noted above, the Benzene court clearly indicated that a risk of one in a thousand could be considered significant and that the Agency would be justified in prescribing reasonable efforts to reduce such a risk.

Therefore, the risk from lung cancer associated with worker exposure to ETS in the workplace meets the Benzene court's characterization of what could be considered significant.

In addition, in evaluating the significance of the risk posed by any particular workplace hazard, the Secretary is entitled to take into consideration not only the rate of risk but the total number of workers exposed to such risk and the absolute magnitude of effects. In this case, evidence in the record shows that approximately between 144 and 722 lung cancer deaths per year are attributable to ETS and that there are presently over 74 million nonsmoking American workers exposed to ETS in their places of employment. On the basis of these data, it would also be reasonable to conclude that Agency action is warranted to reduce this widespread and significant risk, although the Agency would reach this conclusion even without the great magnitude of effects.

As noted above, cancer is not the only serious adverse health effect associated with exposure to ETS. Preliminary estimates indicate that the risk of mortality from heart disease due to ETS exposure is even greater than that of cancer. The Agency estimates that there will be between 2,094 and 13,000 deaths from heart disease per year among nonsmoking American workers exposed to ETS in the workplace. When considered over a working lifetime, this translates into an excess death rate of approximately between 7 and 16 cases of heart disease per thousand attributed to workplace exposure to ETS. Clearly, this risk is significant in itself and combined with the lung cancer risk, the significance of risk is very great.

The proposal seeks to protect nonsmoking employees from the hazards of exposure to ETS in the workplace. It does this by prescribing the conditions under which employees would be allowed to smoke in the workplace, that is, only in separately enclosed designated areas which are separately ventilated. No employee can be required to work in an area where there will be contamination from ETS. This in OSHA's view reduces significant risk to only a small percentage of the current risk. To the extent that there are failures of enforcement of the smoking limitation and of the ventilation system, the risk will not be totally eliminated. Since there is no definition of, nor an established method for quantifying, exposure, it is not possible to determine a "dose limit" that would eliminate significant risk. Even if that were possible, it is not clear it would be the correct policy approach.

29 CFR Part 1990—Identification, Classification and Regulation of Potential Occupational Carcinogens sets forth certain procedures for regulating occupational carcinogens. Those procedures may not allow for the level of public input and policy review that is appropriate for this rulemaking, involving many different types of health effects and a broad range of employers and workers. Accordingly, the Assistant Secretary finds pursuant to 29 CFR Section 1911.4 that "in order to provide greater procedural protections to interested persons or for other good cause consistent with the applicable laws" "it is found necessary or appropriate" to adopt different procedures here.

B. Indoor Air Quality

Poor indoor air quality creates a variety of material impairments of health, two aspects of which are Building-Related Illness and Sick Building Syndrome.

One of the most severe health effects associated with Building-Related Illness is legionellosis, a disease associated with microbial contamination of water sources which is commonly found in the water present in heating and cooling systems of buildings. Legionnaire's disease, caused by the *Legionella* organism, results in pneumonia which is fatal in approximately 20% of the cases. Even when not fatal, it is usually very severe, requiring substantial treatment or hospitalization. As many as 5% of those exposed to *Legionella* will get sick [1]. Legionnaire's disease and other illnesses associated with microbial contamination due to poor indoor air quality are serious health effects that constitute material impairment. Compliance with the indoor air quality provisions set forth in the proposal will substantially reduce these illnesses.

There are numerous other adverse health effects such as nausea, dizziness, fatigue, pulmonary edema, asthma and aggravation of existing cardiovascular disease, which have been associated with poor indoor air quality. Evidence in the record indicates that between 20 and 30% of office buildings are "sick", having environments which may lead to a variety of these effects. Unfortunately, quantitative data are not systematically available on all of these effects.

For purposes of risk evaluation, however, as explained more fully in the risk assessment discussion, the Agency has primarily focussed on two health effects commonly associated with poor indoor air quality: upper respiratory symptoms and severe headaches. The upper respiratory symptoms associated with poor indoor air quality (sick building syndrome) include stuffy nose, runny nose, dry itchy eyes, nose and throat. For purposes of our evaluation, "severe headaches" are defined as those serious enough to require medical attention or restrict activity, but excludes migraines.

Unlike lung cancer and heart disease (health effects associated with exposure to ETS), these effects will not lead to death. There is no doubt, however, that OSHA does have the authority to regulate working conditions that lead to the type of upper respiratory effects and severe headaches described herein.

Clearly the upper respiratory effects and severe headaches associated with poor indoor air quality are of the type that interfere with the performance of work. The severe headaches were such that medical treatment had to be sought; certainly such headaches were impairing at the time they occurred, even though they were not permanent. The upper respiratory symptoms were also severe enough to either require medical attention or restrict activity.

There is ample precedent in OSHA rulemaking proceedings for the regulation of working conditions to avoid health impairments that are material but not life threatening. The Supreme Court in the cotton dust case,[2] concluded that OSHA had the authority to promulgate regulations that would avoid Byssinosis, a respiratory disease which in the large majority of cases is not deadly or disabling, and is reversible if the employee left the cotton mills. Stage ½ byssinosis, the most frequent type, has relatively mild symptoms. In the case of occupational exposure to formaldehyde, the regulation was designed to avoid, among other things, sensory irritation.[3]

Moreover in the "Air Contaminants" standard, OSHA regulated many chemicals, such as acetone, gypsum and limestone which caused less severe impairments of health.[4] In promulgating the final air contaminant rule OSHA analyzed which sorts of conditions would constitute material impairment,

[1] Raw figures from 1992 show approximately 1300 cases of Legionella reported although this is most certainly a gross under-estimation of the scope of the problem, since the disease resembles others and is frequently misdiagnosed.

[2] *AFL-CIO v. Marshall*, 452 U. S. 490 (1981)

[3] See 52 FR 46168, 46235 (12/4/87)

[4] See 54 FR 2332, 2361 (1/19/89)

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the Agency believes that development of written compliance plans are essential to implementation of a successful IAQ program. The written compliance program, inspection and maintenance records, and operator and maintenance schedules which are required to be established under the proposal, are required to be retained under this paragraph. This information essentially documents the desired performance levels of HVAC systems, and the measures necessary to maintain those levels of performance, as well as other measures which should be followed to ensure acceptable indoor air quality. Such data must be available for use by designated persons, current employers, successor employers, and employees as a blueprint for program implementation. Without such data, air quality problems would likely arise due to ignorance of such elements as design occupant densities, equipment schedules, maintenance requirements and frequencies, etc. Records required to be established in response to employee complaints of building-related illness are also required to be retained under this paragraph. Such complaints require the employer to evaluate the need for, and to take if necessary, remedial action to correct observed problems [Ex. 3–1, 3–444B]. Information regarding employee illness is essential in identifying causal factors and trends in adverse health effects. Retention of this health data will aid in the recognition, evaluation and correction of indoor air quality deficiencies which lead to building-related illnesses. Records of building-related illness are proposed to be required to be retained for at least the previous 3 years. OSHA believes that requiring record retention for 3 years of building-related illnesses which occur in nonindustrial environments is reasonable. Such illnesses are not viewed in the same context as industrial illnesses which may be associated with long latency periods, and thus necessitate very long retention periods for health records. Establishment and maintenance of building-related illness records is primarily for the purpose of documenting indoor air quality degradation, so that corrective action can be taken. Requiring records to be retained to preserve a 3 year history of building-related illness, is proposed as being reasonable to aid in the tracking of air quality trends and past experiences [Ex. 3–502].

Other records are also required to be retained for at least the previous 3 years, except to the extent they become obsolete. OSHA does not believe that records such as maintenance and operating schedules which become irrelevant due to HVAC system modification or replacement need be retained further. The records required to be retained under this paragraph must be transferred to successor employers. Since these records contain information specific to the building or facility, as opposed to specific employers, such records should be maintained within affected buildings for future use.

Dates: Paragraph (i)

Paragraph (i) proposes to establish an effective date for this standard of sixty (60) days from publication in the **Federal Register**. A start-up date one year from the effective date is proposed as an adequate period of time for employers to achieve full compliance with all provisions under the rule. The Agency believes that affected employers can develop and implement compliance programs, establish designated smoking areas if smoking is not prohibited, and train employees as proposed under the standard within a one year period from the effective date.

Appendices: Paragraph (j)

The appendices included with this regulation are intended to be informational and, unless otherwise expressly stated in this section, are not intended to create any additional obligations not otherwise imposed, or to detract or reduce any existing obligations.

K. Specific Questions Posed

OSHA solicits data, views and comment on all provisions proposed in this notice. The Agency sets forth questions below to highlight specific areas in the proposal upon which comment is sought.

Regulatory Analysis Issues

(1) Are there any comments on the method used by OSHA to estimate benefits resulting from IAQ provisions of the proposed standard?

(2) Are there studies which document, in quantitative terms, the effectiveness of HVAC maintenance on the decline of indoor air related ailments?

(3) OSHA has estimated a substantial productivity benefit resulting from this proposed standard. What additional studies and other information are available that demonstrate any effect on productivity?

(4) OSHA has preliminarily determined that the direct costs of compliance with this standard will not unduly harm small entities. However, OSHA did not determine how the smoking restrictions in this regulation would affect demand, and therefore profitability, for establishments which provide services and commodities which would be affected by the proposal (e.g., restaurants and bars). OSHA requests comments, including empirical data regarding the demand elasticity of such establishments' patrons who will not be permitted to smoke in the presence of employees.

If economic feasibility is shown to be an issue for establishments such as bars and restaurants, what alternative feasible methods of compliance would prevent workers from being exposed to tobacco smoke?

What other workplaces have circumstances under which provisions of this standard may not be feasible?

(5) During renovation and remodelling, what are the specific elements for implementing control measures to minimize degradation of the IAQ of employees performing such activities and employees in other areas of the building? What are the unit costs associated with the implementation of each control (capital and labor)?

(6) Please describe practices in your workplace by providing answers to the following:

—Describe the business, SIC code number and number of employees in the establishment.

—What type of ventilation systems are presently being used?

—If carbon dioxide monitoring is conducted, how often is it being done and by whom and what are the associated costs?

—Does your establishment have a policy on IAQ? When and why was it implemented? What are the major components? How many employees are affected? What type of costs and cost savings have been associated with such a policy (e.g., operating, maintenance, retrofitting HVAC systems, property damage due to poor IAQ, employee productivity, cleaning, etc.)?

—Is smoking allowed in your establishment? If yes, is it limited to designated smoking areas with separate ventilation?

Scope and Application, Paragraph (a)

(1) Is it necessary and feasible to extend coverage of the entire standard to industrial facilities as well as nonindustrial facilities? Why? Why not? Which provisions lend themselves to application to industrial environments?

(2) Can coverage of the standard feasibly be extended to some industrial facilities but not others? If so, what characteristics distinguish those workplaces in which it is feasible or necessary to apply the standard from those in which it is not?

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production or in the form of increased production and net income for the establishment.

Productivity losses due to indoor air quality may take several forms: employees may be less effective because they feel fatigued or suffer from headaches, eye irritation or other effects. Employees may accomplish less per hour worked or may spend more time away from their work location (e.g., taking breaks or walks outdoor). One company indicated that "since two of my employees have refrained from smoking while working . . ., their production has increased and their overall health seems better to say nothing of the health of those working around them" [Ex. 3-192]. In addition to individual productivity, the quality of indoor air affects organizational productivity such as the visitor and customer satisfaction, impact on sales and revenue and repeat customers.

Little data exist on productivity lost due to poor indoor air quality. A survey of 94 state government office buildings attributes an average productivity loss of 14 minutes per day or 3.0 percent to poor indoor air quality [Ex. 3-1075H2]. Based on information gathered from published resources, the National Energy Management Institute estimates that there is an increase in productivity of 3.5 percent or approximately 15 minutes per day for employees in a building that starts as an unhealthy building, and after IAQ improvements, becomes a healthy building [Ex. 4-240].

To monetize the productivity improvements resulting from implementation of the proposed IAQ standard, OSHA multiplied the average employee payroll by 3.0 percent. As shown in Table VI-7, monetized productivity improvements is estimated at an annual $15 billion.

OSHA requests any studies relating to productivity effects relevant to the proposal be submitted.

(b) Property Damage, Maintenance and Cleaning Costs. High concentrations of contaminants in indoor air can have adverse effects on materials and equipment. Damages may include corrosion of electronic components and electrical current leakage, which may eventually result in equipment malfunction. The costs of materials and equipment damage by indoor air pollutants include maintenance, repair, and/or replacement costs resulting from (1) soiling or deterioration of a materials's appearance, or (2) reduced service life for corroded or degraded appliances, furnishings, and equipment [Ex. 3-1075H2].

Bell Communications Research reported that the seven regional telephone companies have spent large sums ranging from $10,000 to $380,000 per event to replace, clean and repair switches and other electronic equipment malfunctioning as a result of indoor air contaminants.

TABLE VI-7.—AVERAGE ANNUAL COST SAVINGS FROM COMPLIANCE WITH THE IAQ PROPOSED STANDARD DUE TO PRODUCTIVITY GAINS

	Number of employees exposed to poor IAQ	Average annual payroll per employee	Annual productivity[1] improvements (million)
Agriculture, forestry, fishing	83,715	$16,290	$41
Mining	54,210	32,375	*53
Construction	493,125	25,286	374
Manufacturing	1,724,400	28,376	1,468
Transportation	1,023,705	29,655	911
Wholesale and retail trade	4,723,200	20,405	2,891
Finance, insurance, real estate	2,174,445	28,377	1,851
Services	8,077,800	20,811	5,043
Government	2,842,068	32,570	2,777
Total	21,196,668		15,409

[1] Based on productivity loss of 3.0 percent.

Sources: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, 1994. U.S. Department of Labor, Bureau of Labor Statistics. Employment and Wages Annual Averages, 1991. U.S. Bureau of the Census, County Business Patterns, 1990. January 1993.

Microbial contamination can cause significant damage to buildings and equipment and there is anecdotal evidence that damage can be so severe as to make a building unfit for human occupation. OSHA requests comment on the explicit or implicit rental value affected in buildings with such problems.

No quantitative estimates are available on the effects of indoor air on equipment. OSHA requests more information on the effects of indoor air on materials and equipment.

Indoor air pollutants and in particular ETS contribute to increased maintenance and cleaning expenses. Increased maintenance and cleaning costs include: the need to paint walls more frequently, need to clean, repair and replace furniture, upholstery, carpeting and curtains or drapes that have cigarette burns and or odors; the need to wash windows, showcases, and other surfaces that attract ash and dust; and the need to clean ashtrays. A survey of 2,000 companies that had adopted no-smoking policies found that 60 percent of these companies were able to reduce their cleaning and maintenance costs. The savings have been estimated at about $500 per smoker per year (3).

If establishments decide to ban smoking in the workplace, the proposed standard would result in virtually eliminating all smoking related fires, fire fatalities and injuries and direct property damage. Smoking is a leading cause of fire related fatalities. During the 1980's, the National Fire Protection Association reports that smoking materials were the cause of over 200,000 fires per year. This resulted in more than 1,000 civilian fatalities and 3,000 civilian injuries and approximately $300 million in direct property damage. During the period of 1989 to 1990, there was an average of $115 million in direct property damage due to non-residential smoking related fires which resulted in 36 fatalities and 3,212 injuries. OSHA will further investigate this issue and requests available data from the public.

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Table VI–13
Economic Impact of the Proposed Indoor Air Quality Standard

	Total Annualized Costs ($Millions)	Value of Industry Shipments ($Millions)	Pre-tax Profit ($million)	Compliance Cost as a Percent of: Revenue	Profit
AGRICULTURE, FORESTRY, FISHING	$306.1	$121,090	$10,914	0.25%	2.80%
01 Agriculture Production–Crops	$123.6	$45,703	$4,363	0.27%	2.83%
02 Agriculture Production–Livestock	$60.4	$34,770	$2,810	0.17%	2.15%
07 Agricultural Services	$114.8	$35,723	$3,302	0.32%	3.48%
08 Forestry	$3.4	$2,910	$351	0.12%	0.96%
09 Fishing	$4.0	$1,984	$88	0.20%	4.48%
MINING					
13 Oil and Gas Extraction	$26.8	$483,892	$46,190	0.01%	0.06%
CONSTRUCTION (SIC 15, 16 & 17)	$754.7	$546,422	$32,424	0.14%	2.33%
MANUFACTURING	$457.3	$5,078,549	$305,701	0.01%	0.15%
20 Food and Kindred Products	$27.7	$602,970	$29,539	0.00%	0.09%
21 Tobacco Manufacturers	$0.2	$172,271	$19,228	0.00%	0.00%
22 Textile Mill Products	$8.1	$95,923	$5,378	0.01%	0.15%
23 Apparel and Textile Products	$29.9	$92,305	$5,454	0.03%	0.55%
24 Lumber and Wood Products	$45.0	$75,417	$4,152	0.06%	1.09%
25 Furniture and Fixtures	$12.9	$60,540	$3,547	0.02%	0.36%
26 Paper and Allied Products	$8.2	$197,037	$10,946	0.00%	0.08%
27 Printing, Publishing, & Allied Industries	$79.1	$226,709	$17,175	0.03%	0.46%
28 Chemicals & Allied Products	$15.6	$600,941	$36,117	0.00%	0.04%
29 Petroleum Refining and Related Industries	$2.7	$482,927	$24,146	0.00%	0.01%
30 Rubber and Miscellaneous Plastic Products	$18.6	$128,846	$8,134	0.01%	0.23%
31 Leather and Leather Products	$2.6	$24,954	$1,601	0.01%	0.16%
32 Stone, Clay, Glass, & Concrete Products	$19.3	$96,947	$5,827	0.02%	0.33%
33 Metal Industries	$8.6	$187,162	$9,831	0.00%	0.09%
34 Fabricated Metal Products	$41.9	$211,787	$12,515	0.02%	0.33%
35 Machinery, Except Electrical	$66.4	$602,629	$41,393	0.01%	0.16%
36 Electrical Machinery, Equipment & Supplies	$21.1	$296,011	$16,744	0.01%	0.13%
37 Transportation Equipment	$16.1	$641,556	$34,670	0.00%	0.05%
38 Professional & Photographic Equipment & Watches	$14.1	$218,279	$14,442	0.01%	0.10%
39 Misc & Not Specified Manufacturing Industries	$19.2	$63,337	$4,862	0.03%	0.40%
TRANSPORTATION	$286.1	$1,470,658	$157,739	0.02%	0.18%
41 Local and Interurban Transit	$21.2	$24,654	$1,158	0.09%	1.83%
42 Trucking and Warehousing	$134.6	$181,093	$8,049	0.07%	1.67%
44 Water Transportation	$8.3	$44,646	$3,315	0.02%	0.25%
45 Air Transportation	$11.3	$155,387	$7,926	0.01%	0.14%
46 Pipelines, Except Natural Gas	$1.2	$9,819	$3,382	0.01%	0.03%
47 Transportation Services	$49.7	$113,689	$6,890	0.04%	0.72%
48 Communications	$34.4	$414,216	$72,174	0.01%	0.05%
49 Electric, Gas, and Sanitary Services	$25.4	$527,155	$54,845	0.00%	0.05%
WHOLESALE AND RETAIL TRADE	$2,325.9	$4,466,839	$212,377	0.05%	1.10%
50 Wholesale Trade, Durable Goods	$432.2	$1,014,451	$51,235	0.04%	0.84%
51 Wholesale Trade, Nondurable Goods	$256.3	$1,162,472	$48,730	0.02%	0.53%
52 Building Materials and Garden Supplies	$79.5	$107,704	$4,950	0.07%	1.61%
53 General Merchandise Stores	$40.1	$338,896	$18,828	0.01%	0.21%
54 Food Stores	$164.6	$516,213	$18,250	0.04%	1.01%
55 Automobile Dealers and Related Products	$208.8	$490,377	$14,860	0.04%	1.41%
56 Apparel and Accessories Stores	$139.6	$139,673	$11,498	0.10%	1.21%
57 Furniture and Home Furnishing Stores	$122.6	$133,023	$8,398	0.09%	1.46%
58 Eating and Drinking Places	$509.2	$243,437	$13,770	0.21%	3.70%
59 Miscellaneous Retail Stores	$353.0	$320,594	$21,859	0.11%	1.61%
FINANCE, INSURANCE, REAL ESTATE	$618.0	$2,904,557	$86,820	0.02%	0.71%
60 Banking	$78.5	$548,335	NA	0.01%	NA
61 Credit Agencies Other Than Banks	$37.2	$245,431	$32,724	0.02%	0.11%
62 Security and Commodity Brokers and Dealers	$30.9	$228,859	$13,639	0.01%	0.23%
63 Insurance Carriers	$49.0	$1,061,799	NA	0.00%	NA
64 Insurance agents, brokers and service	$135.2	$140,251	NA	0.10%	NA
65 Real Estate, Insurance, Etc.	$263.8	$243,871	$25,619	0.11%	1.03%
67 Holding & other investment offices	$23.5	$136,010	$14,837	0.02%	0.16%
SERVICES	$2,276.9	$2,545,462	$201,141	0.09%	1.13%
70 Hotels and Other Lodging Places	$66.3	$97,230	$7,169	0.07%	0.92%
72 Personal Services	$204.1	$49,263	$5,225	0.41%	3.91%
73 Business Services	$367.1	$452,305	$42,032	0.08%	0.87%
75 Auto Repair, Services, and Garage	$181.6	$103,150	$7,502	0.18%	2.42%
76 Miscellaneous Repair Services	$78.7	$34,536	$3,384	0.23%	2.32%
78 Motion Pictures	$47.1	$74,819	$6,915	0.06%	0.68%
79 Amusement and Recreation, Exc. Motion Pictures	$90.7	$66,954	$4,937	0.14%	1.84%
80 Health Services	$496.3	$592,679	$38,913	0.08%	1.28%
81 Legal Services	$166.0	$85,154	$14,364	0.19%	1.16%
82 Educational Services	$36.1	$418,120	$21,540	0.01%	0.17%
83 Social Services	$128.6	$54,928	$3,301	0.23%	3.90%
84 Museums and Botanical and Zoological Gardens	$3.2	$3,797	$501	0.09%	0.65%
86 Membership Organizations	$111.0	$99,831	$6,050	0.11%	1.84%
87 Engineering, Accounting, Research and Related Services	$290.6	$303,353	$31,273	0.09%	0.93%
89 Miscellaneous Services	$9.4	$74,332	$8,034	0.01%	0.12%
GOVERNMENT	$1,017.2	NA	NA		
	$8,069.13			0.07%	0.94%

Sources: Dun and Bradstreet Market Identifiers data base, 1992.
Insight: Industry Norms and Key Business Ratios, 1992.

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Source: https://www.industrydocuments.ucsf.edu/docs/pxhl0000

TABLE VI–1.—EMPLOYEES WORKING IN BUILDINGS AND OTHER BUILDING CHARACTERISTICS—Continued

Principle building activity	Number of buildings	Percent of all buildings	Total number employees
Part of building	1,634,000	36	

[1] Vacant buildings may contain occupants who are using up to 50 percent of the floorspace.
Source: U.S. Energy Information Administration, Commercial Buildings Characteristics 1989. Washington, DC. June 1991.

The survey also provides information on the number of buildings with heating and air conditioning systems. Total number of heated buildings is estimated to be 3.9 million. Heating systems include boilers, furnaces, individual space heaters, and packaged heating units. Almost one-half of all the buildings are heated by forced-air central systems. Air-distributing heat and cooling systems are most prevalent in office, mercantile and service buildings. The survey reveals that 70 percent of the buildings have air conditioning. It also shows that 80 percent of the buildings have heat and air conditioning, and 12 percent have heat, but no air conditioning.

Over 40 percent of the floorspace built since 1986 was in a building with a computerized energy management and control systems (EMCS). EMCS is an energy conservation feature that uses mini/micro computers, instrumentation, control equipment and software to manage a building's use of energy for heating, ventilation, air conditioning, lighting and/or business related processes. These systems can also manage fire control, safety and security. Overall, EMCS are present in buildings accounting for 23 percent of floorspace. EMCS controls HVAC in only 251,000 buildings or 6 percent of total number of buildings.

However, the DOE survey [Ex. 4–303] does not provide data by two-digit Standard Industrial Classification (SIC). The number of buildings by SIC will determine subsequent costs. OSHA applied the DOE estimates of the number of buildings by type of occupancy (single or multi-tenant) to the number of establishments by two-digit SIC given by the Bureau of Labor Statistics. First, OSHA allocated non-government single tenant buildings (estimated at 3.1 million) across the relative two-digit SIC using the relative two-digit SIC distribution of the number of establishments. Then, OSHA allocated the 0.8 million non-government multi-establishment buildings across two-digit SIC using the relative two-digit SIC distribution of the number of establishments in multi-establishment buildings (2.8 million). All government buildings were considered single tenant buildings. OSHA recognizes that this methodology of classification of buildings by two-digit SIC code may not reflect the fact that establishments in multi-tenant buildings should be allocated across several SICs or the fact that some single establishment buildings may be concentrated in certain SICs instead of all SICs. This is particularly true for the agricultural sector for which farms and farm buildings (silos, grain elevators and barns) are outside the scope of the IAQ portion of the proposal. However, OSHA does not have the data to provide such delineation at this point. Table VI–2 presents OSHA's estimate of the number of buildings by two-digit SIC and by characteristics of occupancy and ventilation system.

TABLE VI–2.—NUMBER OF BUILDINGS AND ESTABLISHMENTS AFFECTED BY IAQ PROPOSED STANDARD

SIC industry	Buildings with single establishments	Buildings with multiple establishments	Total number of buildings	Number of heated buildings	Number of cooled buildings	Number of naturally ventilated buildings [1]
Agriculture, forestry, fishing	136,629	36,557	173,186	147,806	124,312	10,564
Mining	11,976	3,204	15,181	12,956	10,897	926
Construction	336,841	90,127	426,958	364,398	306,475	26,045
Manufacturing	203,995	54,582	258,577	220,684	185,605	15,773
Transportation	127,706	34,170	161,876	138,154	116,193	9,874
Wholesale and retail trade	1,011,035	270,518	1,281,553	1,093,747	919,889	78,175
Finance, insurance, real estate	275,760	73,784	349,544	298,320	250,900	21,322
Services	1,013,057	271,058	1,284,115	1,095,934	921,729	78,331
Government	577,000	.....	577,000	505,000	348,000	35,197
Total	3,694,000	834,000	4,528,000	3,877,000	3,184,000	276,208

[1] Based on estimate of 6.1 percent of floorspace without HVAC.
Source: OSHA, Office of Regulatory Analysis, 1994.

6. Buildings With Indoor Air Problems

Many published reports on building wellness describe buildings in terms of two general categories, sick or well buildings. Some of the published categories, in addition to the terms sick or well are: problem buildings and non-problem buildings, healthy buildings; buildings with high and low rates of IAQ related complaints; sick building syndrome (SBS).

The SBS symptom complex is characterized by a range of symptoms including but not limited to, eye, nose and throat irritation, dryness of mucous membranes and skin, nose bleeds, skin rash, mental fatigue, headache, cough, hoarseness, wheezing, nausea and dizziness [Ex. 4–159]. Within a given building there will usually be some commonality among the symptoms manifested as well as temporal association between occupancy in the building and appearance of symptoms. Many people who work in buildings characterized as having SBS typically exhibit health symptoms that disappear when the person is no longer in the building. In most cases, a physical basis for the occurrence of the SBS can be found: lack of proper maintenance, changes in thermal or contaminant loads imposed during the building's life,

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Table VI–14
Economic Impact of the IAQ Proposed Standard on Affected Small Establishments (19 or fewer Employees)

	Number of Affected Small Establishments	Average Sales Per Establishment	Average Pre-tax Profit Per Small Establishment	Average Annual Cost Per Establishment[1]		Annual Cost Including Designated Smoking Area as a Percent of:		Annual Cost without Designated Smoking Area as a Percent of:	
				with Designated Smoking Area	w/o Designated Smoking Area	Revenue	Profit	Revenue	Profit
AGRICULTURE, FORESTRY, FISHING									
01 Agriculture Production–Crops	102,093	$264,136	$25,213	$2,575	$2,105	0.97%	10.21%	0.80%	8.35%
02 Agriculture Production–Livestock	50,081	$336,726	$27,210	$2,575	$2,105	0.76%	9.46%	0.63%	7.74%
07 Agricultural Services	92,844	$194,763	$18,001	$2,575	$2,105	1.32%	14.30%	1.08%	11.69%
08 Forestry	2,541	$417,923	$46,276	$2,575	$2,105	0.62%	5.57%	0.50%	4.55%
09 Fishing	3,150	$402,260	$16,721	$2,575	$2,105	0.64%	15.40%	0.52%	12.59%
MINING									
13 Oil and Gas Extraction	19,592	$979,674	$93,514	$2,575	$2,105	0.26%	2.75%	0.21%	2.25%
CONSTRUCTION									
15 Building Construction – General Contract	187,408	$614,124	$36,150	$2,575	$2,105	0.42%	6.75%	0.34%	5.52%
16 Heavy Construction other than Building	32,905	$998,944	$57,400	$2,575	$2,105	0.26%	4.49%	0.21%	3.67%
17 Special Trade Contractors	363,437	$289,864	$16,446	$2,575	$2,105	0.89%	13.96%	0.73%	11.41%
MANUFACTURING									
20 Food and Kindred Products	12,715	$2,978,978	$145,940	$2,575	$2,105	0.09%	1.76%	0.07%	1.44%
21 Tobacco Manufacturers	82	$364,228,942	$40,653,837	$2,575	$2,105	*	0.01%	*	*
22 Textile Mill Products	3,142	$3,081,265	$172,736	$2,575	$2,105	0.08%	1.49%	0.07%	1.22%
23 Apparel and Textile Products	15,739	$956,631	$56,646	$2,575	$2,105	0.27%	4.55%	0.22%	3.72%
24 Lumber and Wood Products	30,196	$550,727	$30,318	$2,575	$2,105	0.47%	8.49%	0.38%	6.94%
25 Furniture and Fixtures	7,358	$854,790	$50,079	$2,575	$2,105	0.30%	5.14%	0.25%	4.20%
26 Paper and Allied Products	2,296	$3,996,272	$222,125	$2,575	$2,105	0.06%	1.16%	0.05%	0.95%
27 Printing, Publishing, & Allied Industries	53,420	$504,012	$38,183	$2,575	$2,105	0.51%	6.74%	0.42%	5.51%
28 Chemicals & Allied Products	7,706	$4,056,408	$243,794	$2,575	$2,105	0.06%	1.06%	0.05%	0.86%
29 Petroleum Refining and Related Industries	1,590	$17,849,435	$892,472	$2,575	$2,105	0.01%	0.29%	0.01%	0.24%
30 Rubber and Miscellaneous Plastic Products	7,857	$2,020,670	$127,566	$2,575	$2,105	0.13%	2.02%	0.10%	1.65%
31 Leather and Leather Products	1,292	$1,271,811	$77,882	$2,575	$2,105	0.20%	3.30%	0.17%	2.70%
32 Stone, Clay, Glass, & Concrete Products	11,215	$1,367,705	$82,200	$2,575	$2,105	0.19%	3.13%	0.15%	2.56%
33 Metals Industries	3,247	$5,945,929	$312,311	$2,575	$2,105	0.04%	0.82%	0.04%	0.67%
34 Fabricated Metal Products	22,149	$1,465,387	$86,591	$2,575	$2,105	0.18%	2.97%	0.14%	2.43%
35 Machinery, Except Electrical	40,251	$1,786,536	$122,712	$2,575	$2,105	0.14%	2.10%	0.12%	1.72%
36 Electrical Machinery, Equipment & Supplies	9,819	$2,078,837	$117,591	$2,575	$2,105	0.12%	2.19%	0.10%	1.79%
37 Transportation Equipment	8,008	$3,281,219	$177,318	$2,575	$2,105	0.08%	1.45%	0.06%	1.18%
38 Professional & Photographic Equipment & Watches	7,112	$2,638,370	$174,559	$2,575	$2,105	0.10%	1.48%	0.08%	1.21%
39 Misc & Not Specified Manufacturing Industries	12,861	$500,402	$38,415	$2,575	$2,105	0.51%	6.70%	0.42%	5.48%
TRANSPORTATION									
41 Local and Interurban Transit	13,555	$301,713	$13,269	$2,575	$2,105	0.85%	19.38%	0.70%	15.84%
42 Trucking and Warehousing	97,502	$506,563	$22,514	$2,575	$2,105	0.51%	11.44%	0.42%	9.35%
44 Water Transportation	5,859	$679,634	$65,306	$2,575	$2,105	0.29%	3.94%	0.24%	3.22%
45 Air Transportation	6,465	$2,777,913	$141,702	$2,575	$2,105	0.09%	1.82%	0.08%	1.49%
46 Pipelines, Except Natural Gas	760	$5,877,882	$2,024,804	$2,575	$2,105	0.04%	0.13%	0.04%	0.10%
47 Transportation Services	39,051	$1,144,386	$69,357	$2,575	$2,105	0.23%	3.71%	0.18%	3.04%
48 Communications	19,603	$2,187,022	$381,072	$2,575	$2,105	0.12%	0.68%	0.10%	0.55%
49 Electric, Gas, and Sanitary Services	14,752	$1,998,599	$207,934	$2,575	$2,105	0.13%	1.24%	0.11%	1.01%
WHOLESALE AND RETAIL TRADE				$2,575	$2,105				
50 Wholesale Trade, Durable Goods	317,012	$1,121,613	$56,647	$2,575	$2,105	0.23%	4.55%	0.19%	3.72%
51 Wholesale Trade, Nondurable Goods	183,654	$1,824,535	$76,483	$2,575	$2,105	0.14%	3.37%	0.12%	2.75%
52 Building Materials and Garden Supplies	80,084	$525,197	$24,138	$2,575	$2,105	0.49%	10.67%	0.40%	8.72%
53 General Merchandise Stores	18,794	$892,220	$49,568	$2,575	$2,105	0.29%	5.19%	0.24%	4.25%
54 Food Stores	128,655	$648,393	$22,923	$2,575	$2,105	0.40%	11.23%	0.32%	9.18%
55 Automobile Dealers and Related Products	158,327	$723,265	$21,917	$2,575	$2,105	0.36%	11.75%	0.29%	9.60%
56 Apparel and Accessories Stores	111,066	$277,882	$22,878	$2,575	$2,105	0.93%	11.26%	0.76%	9.20%
57 Furniture and Home Furnishing Stores	86,421	$395,525	$24,970	$2,575	$2,105	0.65%	10.31%	0.53%	8.43%
58 Eating and Drinking Places	276,686	$293,203	$16,565	$2,575	$2,105	0.88%	15.53%	0.72%	12.69%
59 Miscellaneous Retail Stores	281,179	$283,371	$19,321	$2,575	$2,105	0.91%	13.33%	0.74%	10.90%
FINANCE, INSURANCE, REAL ESTATE									
60 Banking	50,543	$1,052,910	NA	$2,575	$2,105	0.24%	NA	0.20%	NA
61 Credit Agencies Other Than Banks	28,447	$1,456,707	$194,494	$2,575	$2,105	0.18%	1.32%	0.14%	1.08%
62 Security and Commodity Brokers and Dealers	22,326	$1,540,044	$91,780	$2,575	$2,105	0.17%	2.81%	0.14%	2.29%
63 Insurance Carriers	32,540	$8,079,913	NA	$2,575	$2,105	0.03%	NA	0.03%	NA
64 Insurance agents, brokers and service	109,303	$476,957	NA	$2,575	$2,105	0.54%	NA	0.44%	NA
65 Real Estate, Insurance, Etc.	212,400	$482,262	$50,664	$2,575	$2,105	0.53%	5.08%	0.44%	4.16%
67 Holding & other investment offices	17,911	$2,598,015	$283,420	$2,575	$2,105	0.10%	0.91%	0.08%	0.74%
SERVICES									
70 Hotels and Other Lodging Places	37,836	$418,657	$30,871	$2,575	$2,105	0.62%	8.34%	0.50%	6.82%
72 Personal Services	165,756	$92,410	$9,801	$2,575	$2,105	2.79%	26.27%	2.28%	21.48%
73 Business Services	266,100	$366,288	$34,039	$2,575	$2,105	0.70%	7.56%	0.57%	6.18%
75 Auto Repair, Services, and Garage	148,515	$232,789	$16,930	$2,575	$2,105	1.11%	15.21%	0.90%	12.43%
76 Miscellaneous Repair Services	63,563	$132,886	$13,020	$2,575	$2,105	1.94%	19.78%	1.58%	16.17%
78 Motion Pictures	36,015	$395,448	$36,549	$2,575	$2,105	0.65%	7.05%	0.53%	5.76%
79 Amusement and Recreation, Etc. Motion Pictures	65,167	$257,247	$19,708	$2,575	$2,105	0.86%	13.07%	0.70%	10.68%
80 Health Services	382,267	$373,803	$24,543	$2,575	$2,105	0.69%	10.49%	0.56%	8.58%
81 Legal Services	133,806	$234,488	$39,555	$2,575	$2,105	1.10%	6.51%	0.90%	5.32%
82 Educational Services	20,847	$644,481	$33,201	$2,575	$2,105	0.40%	7.76%	0.33%	6.34%
83 Social Services	91,062	$137,603	$8,276	$2,575	$2,105	1.87%	31.14%	1.53%	25.45%
84 Museums and Botanical and Zoological Gardens	2,239	$186,414	$20,634	$2,575	$2,105	1.38%	12.48%	1.13%	10.20%
86 Membership Organizations	86,253	$167,648	$10,161	$2,575	$2,105	1.54%	25.34%	1.26%	20.72%
87 Engineering, Accounting, Research & Related Serv.	221,551	$365,644	$35,643	$2,575	$2,105	0.67%	7.22%	0.55%	5.91%
89 Miscellaneous Services	7,643	$130,014	$12,599	$2,575	$2,105	1.98%	20.44%	1.62%	16.71%
						0.52%	7.05%	0.44%	6.02%

Annual compliance cost is based on the following assumptions: establishment floorspace = 10,000 sq. ft., HVAC maintenance cost of $0.21 per sq. ft., designated smoking room cost = $4,000, one maintenance worker will be trained at cost of $8.21, written program development will require one hour of technician time ($15.51/hr.) and 15 minutes of managerial time ($30.84/hr.). Non-recurring costs for designated smoking area are annualized over 20 years at 10% interest rate. All other non-recurring costs are annualized over 10 years at 10% interest rate.
* less than 0.001%
Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, 1994

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health standards for toxic materials or harmful physical agents, to set the standard "* * * that most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity. * * *" It is on the basis of this congressional directive that OSHA has initiated regulatory actions to reduce the adverse health effects associated with occupational exposure to indoor air pollutants.

The discussion below assesses the requisite preconditions for optimal safety in the context of a free market economy, and real world economic factors are compared with the free market paradigm to illustrate the shortcoming of the nonregulatory environment.

(2) Market Imperfections

Economic theory suggests that the need for government regulation is greatly reduced where private markets work efficiently and effectively to allocate health and safety resources. The theory typically assumes perfectly competitive labor markets where employees, having perfect knowledge of job risks and being perfectly mobile among jobs, command wage premiums that fully compensate for any risk of future harm. Thus, theoretically, the costs of occupational injury and illness are borne initially by the firms responsible for the hazardous workplace conditions and ultimately by the consumers who pay for the final goods and services produced by these firms. With all costs internalized, private employers have an incentive to reduce hazards wherever the cost of hazard abatement is less than the total cost to the firm, the work force, and society of the expected injury or illness.

The conditions of perfect competition do not need to be completely satisfied in order for the forces of the market to approximate an efficient outcome. However, some market imperfections can produce sub-optimal results that can be improved upon with regulatory action. In the case of this rulemaking, employees face a significant health risk which is not adequately addressed by current nonregulatory alternatives. OSHA, therefore, believes that it must take appropriate actions to provide greater health protection for workers exposed to toxic substances.

Although OSHA believes that adequate job safety and health could exist in the private market under perfect conditions, the private market often fails to provide acceptable levels of safety and health in instances where these conditions are not met. It appears that at least two of several conditions traditionally considered essential components of perfect markets are absent from the environment in which employees are exposed to hazards associated with exposure to indoor pollutants: (1) Perfect employee knowledge of risks and (2) perfect employee mobility between jobs.

First, evidence on occupational health hazards in general suggests that in the absence of immediate or clear-cut danger, employees and employers have little incentive to seek or provide information on the potential long-term effects of exposure. Employers faced with potentially high compensatory payments may, in fact, have a disincentive to provide information to employees. When relevant information is provided, however, employers and employees might still find informed decisionmaking a difficult task, especially where long latency periods precede the development of chronic disabling disease. Moreover, if signs and symptoms are nonspecific—that is, if an illness could be job-related or could have other causes—employees and employers may not link disease with such occupational exposure.

Second, even if workers were fully informed of the health risks associated with exposure to hazardous substances, many face limited employment options. Nontransferability of occupational skills and high national unemployment rates sharply reduce a worker's expectation of obtaining alternative employment quickly or easily.

In many regions of the country, the practical choice for workers is not between a safe job and a better paying but more hazardous position, but simply between employment and unemployment at the prevailing rates of pay and risk. In addition to the fear of substantial income loss from prolonged periods of unemployment, the high costs of relocation, the reluctance to break family and community ties, and the growth of institutional factors such as pension plans and seniority rights serve to elevate the cost of job transfer. Thus, especially where wages are more responsive to the demands of more mobile workers who tend to be younger and perhaps less aware of job risks, hazard premiums for the average worker will not be fully compensated. Where this is the case, labor market negotiations are unlikely to reflect accurately the value that workers place on health.

In addition to these market imperfections, externalities occur if employers and employees settle for an inefficiently low level of protection from hazardous substances. For the competitive market to function efficiently, only workers and their employers should be affected by the level of safety and health provided in market transactions. In the case of occupational safety and health, however, society shares part of the financial burden of occupationally induced diseases, including the costs of premature death, chronic illness, and disability. Those individuals who suffer from occupationally related illnesses are cared for and compensated by society through taxpayer support of social programs, including welfare, Social Security, and Medicare.

If private employers do not have to pay the full cost of production, they have no economic incentive to reduce hazards whenever the cost of hazard abatement is greater than the cost of the expected illness. In this way, the private market fails to produce optimal levels of safety.

(3) Alternative Non-regulatory Options

Based on the above evidence, OSHA has concluded that the private market has failed to provide optimal levels of safety to employees. Consequently, some form of intervention that fosters safer work environments must be used to reduce occupational exposure. Because such intervention need not occur through government regulation, OSHA has considered the effectiveness of other non-regulatory options: (1) relying on tort litigation and (2) relying on workers' compensation programs.

(a) Tort Liability. The use of liability under tort law is one nonregulatory alternative that has been increasingly used in litigation concerning occupationally related illnesses. Prosser [Ex. 4–256] describes a tort, in part, as a "civil wrong, other than a breach of contract, for which the court will provide a remedy in the form of an action for damages".

If the tort system applies, it would allow a worker whose health has been adversely affected by occupational exposure to a hazardous substance to sue and recover damages from the employer. Thus, if the tort system is effectively applied, it might shift the liability of direct costs of occupational disease from the worker to the firm under certain specific circumstances.

With very limited exceptions, however, the tort system is not a viable alternative in dealings between employees and employers. All states have legislation providing that Workers' Compensation is either the exclusive or principal remedy available to employees against their employers. Thus, under tort law, workers with an occupational disease caused by exposure to a

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Source: https://www.industrydocuments.ucsf.edu/docs/pxhl0000

No data are available as to the number of employees exposed to poor indoor air quality. Based on OSHA's percentage of problem buildings (30 percent), OSHA assumed that 30 percent of employees working indoors are exposed to poor indoor air quality. Therefore, the number of employees potentially affected is 21 million.

8. Environmental Tobacco Smoke

Environmental Tobacco Smoke (ETS) represents one of the strongest sources of indoor air contaminants in buildings where smoking is permitted. ETS is a mixture of irritating gases and carcinogenic tar particles and is considered one of the most widespread and harmful indoor air pollutants.

(a) Smoking ordinances[8] and policies. State and Local Governments have adopted an increasing number of ordinances and regulations limiting smoking in public and private worksites. The restrictiveness of these laws varies from simple, limited prohibitions to laws that ban smoking. Forty-five states and the District of Columbia restrict smoking in public workplaces and 19 states and the District of Columbia restrict smoking in private workplaces.

There are 397 city and county smoking ordinances covering 22 percent of the total population [Ex. 4–305]. A total of 297 cities and counties mandate the adoption of workplace smoking policies. Typically these provisions require employers (private and public) to maintain a written smoking policy. Ordinances range from requirements for written smoking policies to the total elimination of smoking in the workplace. A total of 505 cities and counties limit smoking, specifically in restaurants. The requirements range from a nonsmoking section of unspecified size to the banning of all smoking [Ex. 4–305].

A 1991 survey of company smoking policies shows that of the 85 percent of firms with smoking policies, 34 percent have complete bans and another 34 percent prohibit smoking in all open work areas. Over 90 percent of non-manufacturing establishments have smoking policies [H–030 Ex. 77].

Workplace smoking policies are more common in larger businesses. In a survey of personnel managers, 63 percent of those with 1,000 or more employees reported having a smoking policy compared with 52 percent of companies with fewer employees. In the same survey, smaller companies were half as likely as larger ones to have a policy under consideration. Similar findings were reported by the National Survey of Worksite Health Promotion Activities, in which larger worksites were more likely than smaller ones to report smoking control activities. In a survey of private New York city businesses, only 4 percent of companies with fewer than 100 employees had a written smoking policy [Ex. 3–1030Q].

(b) Number of nonsmokers working indoors. Based on the National Health Interview Survey, OSHA estimated that 74.2 million employees or 73.01 percent of the U.S. labor force covered by OSHA are nonsmokers. Table VI–4 presents the distribution of nonsmoking employees by two digit SIC.

Results of population based surveys show that 88 percent of nonsmokers are aware of the negative health consequences of ETS. Despite this general awareness, exposure to ETS is pervasive [Ex. 4–98]. To determine the occupational exposure of nonsmoking employees to ETS, OSHA used the estimate provided by the 1991 National Health Interview Survey. The survey, requested information from employed individuals on whether during the past two weeks anyone smoked in their immediate work area. Based on results adjusted for non-response and weighted to reflect national estimates, 18.81 percent reported exposure to ETS. OSHA believes that the 18.8 percent is an underestimate since it is based solely on self reported information and the question was not very specific in defining "immediate" work area. A recent reanalysis of a study by Cummings et al. [Ex. 4–68] shows that 48.67 percent of currently employed nonsmokers reported ETS exposure at work and not at home [Ex. 3–442F].

TABLE VI–4.—EMPLOYEES EXPOSED TO ENVIRONMENTAL TOBACCO SMOKE

SIC industry	Nonsmoker employees[1]	Number of employees exposed to ETS	
		Lower bound (18.81%)	Upper bound (48.67%)
Agriculture, forestry, fishing	1,008,007	189,606	490,597
Mining	249,256	46,885	121,313
Construction	3,479,876	654,565	1,693,655
Manufacturing	13,050,099	2,454,724	6,351,483
Transportation	3,953,337	743,623	1,924,089
Wholesale and retail trade	19,041,884	3,581,778	9,267,685
Finance, insurance, real estate	3,995,180	751,493	1,944,454
Services	21,687,986	4,079,510	10,555,543
Government	7,735,393	1,455,027	3,764,816
Total	74,201,019	13,957,212	36,113,636

[1] Based on 73.01 percent nonsmoking employees.
Source: OSHA, Office of Regulatory Analysis, 1994.

By applying the lower and upper ranges of exposure, OSHA estimates that the number of nonsmoking employees exposed to ETS to be 13.9 to 36.1 million employees.

C. Nonregulatory Alternatives

(1) Introduction

The declared purpose of the Occupational Safety and Health (OSH) Act of 1970 is "* * * to assure so far as possible every working man and woman in the Nation safe and healthful working conditions and to preserve our human resources. * * *" Thus, the Act requires the Secretary of Labor, when promulgating occupational safety and

[8] A smoking ordinance may mean any local law which addresses public smoking in some fashion to protect non-smokers.

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Source: https://www.industrydocuments.ucsf.edu/docs/pxhl0000

TABLE VI–12.—OPTIONAL COST FOR PROVIDING SEPARATE SMOKING AREAS—Continued

	Number of establishments providing designated smoking areas [1]		Annualized first-year cost [2] ($ million)
	In single establishment buildings	In multi-establishment buildings	
Transportation	34	7	0.019
Wholesale and retail trade	93,411	36,058	60.829
Finance, insurance, real estate	83	16	0.046
Services	11,188	3,968	7.121
Total	104,932	40,091	68.138

[1] Number of establishments adjusted for percentage banning smoking as follows: Manufacturing 23%; Transportation and Utilities 36%; Wholesale and Retail 7%; average rate for all other industries 25%. Number of establishments included represent 50 percent of large establishments in buildings with 3 or more floors and with floor space greater than 100,000 sq. ft. Number of establishments include 50% of all establishments in SIC 58 (Eating and Drinking places) and 70 (Hotels).

[2] Cost for making ventilation changes is estimated at $4,000/smoking room which accommodates up to 10 smokers. Initial costs are annualized over 20 years at a 10 percent interest rate.

Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, 1994.

More than half of the buildings sampled were occupied during renovation activities. However, all projects in which asbestos-related work was being performed were sealed off from the building occupants. A variety of renovation projects were performed in buildings ranging in project area from 15 to 900,000 square feet, in duration from one to 156 weeks and in cost from $700 to more than $10 million. The average cost was approximately $0.3 million and the average duration for a project was 13 weeks.

However, no data are available on the cost to provide controls required under the proposed IAQ standard or for current industry compliance for chemical exposure other than exposure to asbestos. OSHA assumed minimal cost due to the nature of these processes.

F. Economic Impact and Regulatory Flexibility Analysis

The previous section presented the costs to all industries of complying with the proposed standard. This chapter examines projected economic and environmental impacts on those industries. OSHA developed quantitative estimates of the economic impact of the proposed standard on the affected industries. Data on profits are presented to illustrate the scale of profitability of affected industries and do not necessarily represent their ability to pay for proposed standard provisions.

OSHA assessed the potential economic impacts and has preliminarily determined that the standard is economically feasible for each of the major industry groups that will be affected. OSHA conducted its analysis at the two digit SIC level. This has been OSHA's procedure for doing regulatory impact analyses for other proposed standards. OSHA preliminarily concludes that this is reflective of the actual impact on the average firm within each subsector. It does not appear that the affected groups will experience significant adverse economic impact as a result of the standard. However, if any interested person has information to show that the analysis at the two digit level is not representative of the potential economic impact of the proposal, OSHA requests the following information: Reasons why the preliminary regulatory analysis is not reflective of the actual anticipated costs in any particular sector; specific information as to why the analysis at the two digit level fails to adequately represent the economic impact; and specific information to help OSHA to better predict the impact on the sector in question. Such information should be included in the comments on the proposal.

In accordance with the Regulatory Flexibility Act of 1980, OSHA additionally examined the potential for an unduly burdensome impact on small entities. OSHA believes that the standard will not have significant adverse effect on a substantial number of small entities. However, OSHA requests comment on those workplaces for which compliance with the proposed standard would prove economically and technologically challenging (e.g., restaurants, bars and other "public" places where employees are exposed to customer's tobacco smoke). While it is technologically feasible to ban smoking in those establishments, there may be other countervailing problems, legal and economic, which OSHA should consider.

(1) Economic Feasibility

In order to determine the economic feasibility of the rule, OSHA compared estimated compliance costs with: (1) The value of sales and (2) before-tax profits. All financial data developed for this analysis are based on information from Dun and Bradstreet's annual credit survey. Aggregate sales data for 1991 were taken from the D&B Market Identifiers data base [Exs. 4–94, 4–95, 4–96]. Mean profit rates (profit as a percentage of sales) were taken from D&B's Insight data base; OSHA averaged data for 1990, 1991 and 1992.[11]

Using a conversion formula[12] based on the federal corporate tax schedule, OSHA calculated pre-tax profits from its estimate for post-tax profits. It should be noted that the sales and profit data, while the most recent available, reflect conditions during a cyclical trough; therefore, impacts may depict a worst case scenario. In the case of the federal government sector, price increases for services rendered may not apply. Budgets are usually fixed (in the short run) and compliance costs are paid by reducing funds for other items in the budget.

Where industry enjoys an inelastic demand for its product, an increase in operating costs can ordinarily be passed

[11] Dun's Insight computer data base presents data from their three most recent annual industry Norms and Key Business Ratios publications. For most industry groups, OSHA averaged data for three years.

[12] This conversion implicitly assumes individual business establishments are separate corporations. Because more than one establishment may be grouped together for tax purposes, the conversion will tend to underestimate pre-tax profits. State, local and other business taxes have not been factored into the conversion formula. Additionally, because average tax rates may decline as pre-tax profits decline, the after-tax impact to the company may be less than suggested here.

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on a control device or centralized control system. Depending on the condition of the HVAC equipment, inspection and maintenance may include simple housekeeping of equipment and air transport pathways and/or catastrophic failure maintenance to repair/replace failed equipment. Also, there may be cases where a number of buildings will require major changes in the HVAC system such as enlarging the size of the outside air intake.

The cost for providing maintenance first requires an estimate of the number of buildings without regular HVAC maintenance. The 1989 Commercial Buildings Characteristics survey by the Department of Energy estimates that 46 percent of the buildings have regular HVAC maintenance. Therefore, the total number of buildings requiring maintenance is estimated at 2.3 million. OSHA then determined the number of problem buildings without HVAC maintenance by applying the OSHA estimate of 30 percent (presented in section B). The number of problem buildings without HVAC maintenance is estimated at 0.7 million.

In general, the average cost per year to maintain a commercial HVAC system is a function of a number of factors. These factors include the type of system, the age of the system, the size of the system, layout of the system, reliability of the equipment installed. In addition to the physical characteristics of the system, the cost per year to maintain the system also depends on the operation of the system, the maintenance policies of the owner, the skill levels of the operating engineers and maintenance workers, and whether the maintenance is carried out by employees of the building owner or is the responsibility of an outside company.

Bank of America's maintenance costs for its 2,000 worksites averaged $4 million per year or an average of $2,000 per worksite [Ex. 3–552]. One facility, a high-rise office building, reported an annual cost of approximately $0.6 million [Ex. 3–448]. DOW Chemical Company's estimate for ventilation systems maintenance ranges from $0.17 to $0.25/sq.ft/yr [Ex. 3–502]. Therefore, OSHA used an average of $0.21/sq.ft/yr to compute the cost of HVAC maintenance.

In addition to regular HVAC maintenance, buildings with known IAQ problems will require other improvements such as (1) relocating air intakes and other pathways of building entry to restrict the entry of outdoor air contaminants, or (2) installing local source capture exhaust ventilation or substitution within workspaces where air contaminants are being emitted, or (3) increasing ventilation effectiveness, or (4) reduce unwanted infiltration, or (5) monitor outside air quantity to meet ventilation requirements. The National Energy Management Institute developed a cost model for implementing IAQ improvements which is based on the distribution of buildings with IAQ problems by climate zone, building activity and size, and characteristics of ventilation systems. The average cost to implement the actions listed above are estimated to be $1.14 per square foot. These improvements will only be required for the initial year.

The cost equation for implementing the compliance program is as follows:

$$C_p = M_s (N_h \times C_a + N_p \times C_i \times A_{20})$$

where

C_p=cost for providing regular HVAC maintenance

M_s=mean square footage per building (14,000)

N_h=number of buildings without HVAC maintenance

C_a=cost per square foot for providing HVAC maintenance ($0.21)

N_p=number of problem buildings without HVAC maintenance

C_i=cost per square foot for providing HVAC maintenance and IAQ improvement actions ($1.14)

A_{20}=Annualization factor at 10% over 20 years (0.117)

Non-recurring first year costs were annualized over 20 years at 10 percent interest rate. As presented in Table VI–10, the annualized first-year cost is estimated at $1.3 billion. OSHA anticipates total annual costs of $8.0 billion.

(c) Training for HVAC Maintenance Workers and Informing Employees About the Indoor Air Quality Standard. The proposed standard requires training for all building maintenance workers involved in building system operation and maintenance. Standards of maintenance vary dramatically in the HVAC industry and sometimes are deficient where untrained personnel are designated to maintain very complex systems.

Training programs for workers must include at least information on: (1) How to maintain adequate ventilation of air contaminants generated during building cleaning and maintenance, and (2) how to minimize adverse effects on indoor air quality during the use and disposal of chemicals and other agents.

The exact cost of training will vary among establishments depending on whether employees are trained in-house or sent to outside training programs or consultants. OSHA estimated the costs for the trainer who must research, prepare and direct the sessions. For the time involved in the training session, a range of costs for the instructor could be developed. For example, the wage costs for the trainer could represent from 50 percent of the trainee labor costs (if there are only two in the class) to 5 percent if there are 20 trainees in a class. For the preparation time, OSHA judged that the trainer will require a special study seminar, such as that taught by the Building Owners and Managers Association International.

TABLE VI–10.—COST FOR SYSTEM OPERATION AND MAINTENANCE PROVISION

	Buildings with IAQ problems and without HVAC maintenance			Buildings without HVAC maintenance and without IAQ problems		Total annual cost ($ million)
	No. of buildings with HVAC	Annualized[1] cost to improve IAQ ($ million)	Annual[2] cost ($ million)	No. of buildings	Annual cost[3] ($ million)	
Agriculture, forestry, fishing	26,139	$49	$77	60,990	$179	$305
Mining	2,291	4	7	5,346	16	27
Construction	64,442	121	189	150,364	442	752
Manufacturing	39,027	73	115	91,062	268	456
Transportation	24,432	46	72	57,007	168	285
Wholesale and retail trade	193,423	363	569	451,320	1,327	2,258
Finance, insurance, real estate	52,756	99	155	123,098	362	616
Services	193,810	363	570	452,222	1,330	2,263

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Source: https://www.industrydocuments.ucsf.edu/docs/pxhl0000

TABLE VI–5.—CASES AND CASES AVOIDED OF OCCUPATIONALLY DEVELOPED UPPER RESPIRATORY SYMPTOMS AND HEADACHES IN BUILDINGS WITH HVAC SYSTEMS OVER A WORKING LIFETIME OF 45 YEARS

	Headaches		Upper respiratory symptoms	
	Baseline cases[1]	Cases avoided due to IAQ standard	Baseline cases[2]	Cases avoided due to IAQ standard
Agriculture, forestry, fishing	14,936	11,948	22,272	17,818
Mining	9,672	7,737	14,423	11,538
Construction	87,978	70,383	131,196	104,957
Manufacturing	307,650	246,120	458,777	367,021
Transportation	182,639	146,111	272,357	217,885
Wholesale and retail trade	842,666	674,133	1,256,607	1,005,286
Finance, insurance, real estate	387,943	310,354	578,511	462,809
Services	1,441,160	1,152,928	2,149,099	1,719,279
Government	507,053	405,643	756,132	604,906
Total	3,781,698	3,025,358	5,639,374	4,511,499

[1] Based on OSHA estimate of occupational headache risk of 57 per 1,000 employees over a working lifetime of 45 years.
[2] Based on OSHA estimate of occupational upper respiratory symptoms risk of 85 per 1000 employees over a working lifetime of 45 years. OSHA estimate for cases prevented through proposed standard is 80 percent.
Source: OSHA, Office of Regulatory Analysis, 1994.

OSHA's estimates are based upon the exposure profile (presented in Table VI–3) and OSHA's quantitative risk assessment (discussed in detail in the preamble to the proposal). The OSHA estimates of lifetime risk of death attributable to exposure to ETS in the workplace range between 0.4 and 1 for lung cancer and between 7 and 16 for coronary heart disease, per 1,000 exposed employees. OSHA's estimate of the attributable risks suggest that all baseline cases of lung cancer and coronary heart disease will be prevented due to elimination of exposure of nonsmokers to ETS in the workplace.

Table VI–6 presents estimates of the incidence of work-related cases avoided of lung cancer and heart disease following either the banning of smoking in the workplace or limiting smoking to designated smoking areas. OSHA estimates that approximately between 5,583 and 32,502 cancer deaths and 97,700 to 577,818 coronary heart disease deaths related to occupational exposure to ETS will be prevented over the next 45 years. This represents 140 to 722 cancer deaths per year and 2,094 to 13,001 heart disease deaths per year.

3. Costs Savings

OSHA has also preliminarily determined that the estimated number of deaths or illnesses prevented understates the actual benefits that would occur under the proposed standard. Significant additional economic benefits, apart from the lives saved and illnesses averted, are anticipated most of which can not be quantified at this time.

TABLE VI–6.—CASES AVOIDED OF OCCUPATIONALLY DEVELOPED LUNG CANCER AND CORONARY HEART DISEASE PER EMPLOYEES EXPOSED TO ETS OVER A WORKING LIFETIME OF 45 YEARS

	Number of non-smoking employees exposed to ETS at work		Coronary heart[1] disease avoided		Lung cancer[2] deaths avoided	
	Lower bound	Upper bound	Lower bound	Upper bound	Lower bound	Upper bound
Agriculture, forestry, fishing	189,606	490,597	1,327	7,850	76	442
Mining	46,885	121,313	328	1,941	19	109
Construction	654,565	1,693,655	4,582	27,098	262	1,524
Manufacturing	2,454,724	6,351,483	17,183	101,624	982	5,716
Transportation	743,623	1,924,089	5,205	30,785	297	1,732
Wholesale and retail trade	3,581,778	9,267,685	25,072	148,283	1,433	8,341
Finance, insurance, real estate	751,493	1,944,454	5,260	31,111	301	1,750
Services	4,079,510	10,555,543	28,557	168,889	1,632	9,500
Government	1,455,027	3,764,816	10,185	60,237	582	3,388
Total	13,957,212	36,113,536	97,700	577,818	5,583	32,502

[1] OSHA estimate of occupational coronary heart disease risk for lower and upper bound exposure of 7 to 16 per 1,000 employees over a working life of 45 years.
[2] OSHA estimate of occupational lung cancer risk for lower and upper bound exposure of 0.4 to 0.9 per 1,000 employees over a working life of 45 years.
Source: OSHA, Office of Regulatory Analysis, 1994.

The major forms of these savings are efficiency and productivity improvements, cost reductions in operations and maintenance, and reduced incidence of property damage.

(a) Worker Productivity. Productivity gains are realized when less labor input is required per unit of production. A productivity gain can, therefore, take the form of either a decrease in the labor hours needed to maintain the level of

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constructed, renovated, or remodelled, whichever was most recent [Ex. 3–18]. Paragraph (d)(2) proposes to require employers to conduct building system inspection and necessary maintenance activities as often as necessary to reduce the likelihood of indoor air quality problems related to the building systems [Ex. 3–26]. Further requirements under paragraph (d) are: Assure that the HVAC system is operable during all work shifts, (d)(3) [Exs. 3–56, 3–226, 3–347, 3–436]; implement the use of general or local ventilation where maintenance activities may result in hazardous chemical or particulate exposures in other areas of the building, (d)(4) [Exs. 3–347, 3–502]; maintain relative humidity below 60% in buildings with mechanical cooling systems, (d)(5) [Exs. 3–34, 3–61, 3–505B]; during regular maintenance, as described in subparagraph (d)(1), measure carbon dioxide levels. When they exceed 800 ppm, check to make sure the HVAC system is operating as it should and correct deficiencies if necessary, (d)(6) [Exs. 3–10, 3–34, 3–214]; assure that buildings without mechanical ventilation are maintained so that windows, doors, vents, etc., designed or used for natural ventilation are in operable condition, (d)(7); assure that mechanical equipment rooms and any non-ducted air plenums or chases are maintained in a clean condition, free of hazardous substances, and asbestos, if friable, is encapsulated or removed so that it does not enter the air distribution system, (d)(8) [Exs. 3–29, 3–500]; assure that inspections and maintenance of the HVAC system are performed by or under the direction of the designated person, (d)(9) [Ex. 3–29]; establish a record of HVAC system inspections and maintenance, (d)(10) [Ex. 3–26]; assure that employees performing work on HVAC systems are provided with and use appropriate personal protective equipment, (d)(11) [Ex. 3–56]; evaluate the need to perform alterations of the HVAC system in response to employee reports of building-related illness, (d)(12); and take such remedial measures as the evaluation shows to be necessary, (d)(13).

OSHA believes that implementation of each of the actions prescribed in this proposed paragraph are integral elements in the indoor air quality program. Provisions which address inspection, maintenance, alteration, and operation of building systems are believed to be essential to ensure successful functioning of system functioning.

Paragraph (d)(1) proposes to require that employers operate and maintain the HVAC system to provide at least the minimum outside air ventilation rate. Available evidence in the literature supports this requirement. The literature which supports the case for ventilation with outside air falls into two categories. One category includes case studies which are generated when IAQ complaints require on-site responses and the investigators report their findings through IAQ forums sponsored by professional organizations like the American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc. and the American Industrial Hygiene Association. These studies report that the lack of outside ventilation air resulting from operational or maintenance deficiencies as one of the causes of IAQ complaints. Many of the studies include abatement recommendations to ventilate with outside air as feasible per the original design intent. The second category includes research projects which also support the case for ventilating buildings with at least the recommended minimum of outside air. Research in the areas of ventilation efficiency, tracer gas analysis, dilution/removal of internally generated contaminants, and environmental perceptions mostly support this contention.

All three major building codes in the United States which are used in the design of new and retrofitted facilities mandate minimum outside air ventilation rates in mechanically-ventilated buildings. These three code bodies include the Building Officials and Code Administrators International, Inc. (BOCA), the International Conference of Building Officials (ICBO), and the Southern Building Code Congress International, Inc. (SBCCI). Per Section 102 of the 1991 Uniform Building Code as promulgated by the ICBO, the purpose of the building code is "to offer minimum standards to safeguard life or limb, health, property and public welfare by regulating and controlling the design, construction, quality of materials, use and occupancy * * *". Clearly, there is a significant commitment of resources by these code bodies to offer design guidance through the building codes to designers to insure that a facility is capable of delivering a minimum amount of outside air to its' occupants. This concept is supported by the efforts of plan reviewers and building inspectors in local governmental jurisdictions throughout the United States who ensure that facilities are constructed per the building codes. Considering the up-front efforts of these code officials, designers, and construction teams, it is reasonable from a standpoint of continuity, to require that buildings be operated and maintained to the same design intent.

This provision is not meant to require rebuilding or retrofitting HVAC systems in response to minor work. For example, such steps would not be required for any renovation work that does not modify the building's configuration or the conditions that would be affected by the building code applicable at the time the system was installed or last modified.

As part of maintenance, there should be a predictive element which periodically checks the HVAC system to evaluate conformance with paragraph (d). This check should conform with the proposals of paragraph (d)(2) which requires an inspection and maintenance of the building systems. This periodic visual inspection is focused by the checklist outlined in paragraph (c) and targets those components that directly impact indoor air quality. In the field of occupational safety and health, as practiced by industrial hygienists, it is common practice to perform walk-around inspections. On the other hand, the HVAC industry often relies heavily on remote sensing to characterize system performance. Therefore, this required visual inspection will help identify those deficiencies that would otherwise be missed, such as microbial contamination in cooling coil condensate drip pans.

Paragraph (d)(3) requires that the facility HVAC system is operating during all workshifts. The employer must provide the minimum outside ventilation rate for contaminant dilution and removal whenever the building is occupied and used. OSHA understands that the minimum outside air ventilation rate may in practice only be provided when the building is fully occupied or utilized. It is not uncommon for office buildings to be occupied from 6 a.m. to 7 p.m. to accommodate flexible work schedules but the HVAC system may only be in operation from 8 a.m. to 5 p.m. to conserve energy. The technical rationale for this strategy is typically based on the recommendations of the American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc. (ASHRAE) in their Standard 62–1989 titled "Ventilation for Acceptable Indoor Air Quality". Section 6.1.3.4 and Appendix "G" of ASHRAE Standard 62–1989 [Ex. 4–333] offers a rationale for the lead/lag operation of ventilation systems to accommodate transient occupancy. The basis for the rationale is that there is capacity in air to dilute contaminants if the space has been previously unoccupied for several

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properly, and the taking of remedial action where warranted. Building-related illnesses are often potentially severe and are often traceable to a specific contaminant source such as ETS, microbial growth, and a host of other chemical or biologic substances which must be attended to mitigate degradation of indoor air quality.

The term "Designated person" means a person who has been given the responsibility by the employer to take necessary measures to assure compliance with this section and who is knowledgeable in the requirements of this standard and the specific HVAC system servicing the affected building or office. As noted above a "Designated person" must be knowledgeable in HVAC system functioning. Provisions in the standard propose to require the "Designated person" to oversee the establishment and implementation of the IAQ compliance program, and oversee building systems inspection and maintenance activities, thus this person must have technical expertise in those areas. OSHA believes that there is a need for central responsibility in affected buildings and facilities [Exs. 3–434, 3–444b, 3–507]. Of course OSHA recognizes that in certain circumstances the "Designated person" may merely supervise or coordinate the activities of outside contractors or shift-workers who have responsibility for maintaining parts of the building systems. Building systems and other factors affecting indoor air quality are sufficiently complex and unique to suggest the necessity of appointing a designated person who is on site to act on the employers behalf in this regard. For example, multiple employers may be engaging in different activities within a facility that affect building system functioning or air quality and actions by one employer may subject employees of other employers to environmental hazards. Fragmentation of responsibility and lack of communication has been observed by OSHA in the nonindustrial workplace. For example, when responding to an indoor air quality/building-related illness complaint, the OSHA Compliance Officer may need to gather information from a number of responsible facility groups like tenant leasing, facilities engineering, housekeeping, maintenance, operations, and energy management. These diverse groups may have little or no central authority and direction especially if they are outside contractors. The designated person would be in a position to mitigate the consequences of such diversity by being aware and responsible for the overall environmental conditions in the building or facility.

Other OSHA health standards have adopted similar requirements with respect to those proposed in this Notice regarding the designated person. For example, final standards for chromium (57 FR 42102) and lead (58 FR 26590) require that a technically knowledgeable "competent person" be on site during construction activities, which often involve multiple employers. OSHA concluded in those standards that designating a person to act on the employers' behalf to ensure compliance with various provisions of those standards, was necessary because of the need for continual site characterization and analysis to identify the hazards present and the types of control measures and remedial actions that are effective. For these same reasons, OSHA proposes requirements for designated persons under this notice.

The term "Designated smoking area" means a room in which smoking of tobacco products is permitted. The Agency believes that establishment of "designated smoking areas" is necessary to prevent employee exposure to ETS in workplaces where smoking is not prohibited. Provisions are included in this proposal addressing design, construction and operation of such areas to meet this purpose.

The term "Director" means the Director, National Institute for Occupational Safety and Health (NIOSH), U.S. Department of Health and Human Services, or designee.

The term "Employer" means all persons defined as employers by section 3(5) of the Occupational Safety and Health Act of 1970 including employers (such as building owners or lessees) who control the ventilation or maintenance of premises where employees of other employers work. For purposes of the proposal, an employer is also defined as a person who exercises control over the ventilation systems in the workplace. Control over the ventilation systems is a multi-faceted concept: it includes maintenance, recordkeeping and the development and implementation of the indoor air quality compliance plan. While responsibility for various aspects of the ventilation system encompasses many duties, the proposal does not necessarily contemplate that all of the duties will be performed by the same person. The proposal is flexible in that regard and responsibility for the various aspects can be shared by various persons depending on the circumstances.

In many instances the employer will either be the owner of the building where the workplace is located or will be a long term lessee, responsible under the lease for the care and maintenance of the property. In these cases, the owner/employer would take care of the ventilation system by designating knowledgeable persons within his employ to the necessary tasks or by hiring competent contractors.

In other cases, there will be a number of different businesses all located in separate leased space within the same building. In these instances the various employer/lessees would probably share responsibility for compliance with the proposed standard. For example, each individual lessee might be obligated to provide the building owner with a description of the work activity planned for within its particular leased space, including the number of employees or visitors expected, the hours of work operation and any situations where air contaminants may be released into the workplace air. Air contaminants might reasonably be expected to be released into the workplace air as a result of the installation of new furniture or wall coverings, any painting or remodeling scheduled to take place or any pest extermination activity within the premises. Each employer would, of course, be responsible for reporting to whoever is in charge of the ventilation system, any employee complaints or signs or symptoms that may be related to building-related illness.

The building owners or whoever is in charge of the maintenance of the ventilation system would be in a position to develop standard operating procedures for the building systems as well as special procedures for emergencies and maintenance. In addition such a person would be in a position to know or develop an appropriate maintenance schedule and to gather relevant documents to assist in the care and maintenance of the ventilation system, such as diagrams of the system, manufacturers manuals, and inspection guidelines and schedules for the proper maintenance of such systems. The same person might also be responsible for maintaining and operating the HVAC system to provide the required air ventilation rate and desired relative humidity.

The proposal is designed in this performance oriented manner to afford affected employers the flexibility to assure the establishment and maintenance of a system to provide healthful indoor air quality in the most sensible and efficient way possible considering their particular circumstances.

The Occupational Safety and Health Act gives the Secretary the right to

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substances. Written plans provide information to allow OSHA, the employer, and employees to examine the control methods chosen and evaluate the extent to which these planned controls are being implemented.

Paragraph (c)(3) proposes to require the employer to establish written plans for compliance. Specifically, paragraphs (c)(3)(i), (c)(3)(ii), and (c)(3)(iii) propose to require general, descriptive information about: the facility, building systems, building function and building use patterns. This general building description is believed to be essential information of a building profile which is necessary for a basic understanding of the building systems and which is necessary to set the foundation for the operations and maintenance information required in other paragraphs.

Further, in paragraph (c)(3)(iv), OSHA believes that it is necessary to require written information which describes daily operation and management of the facility building systems which directly affects IAQ. When it comes to operations and management, organizational fragmentation within nonindustrial buildings may be further exacerbated by the lack of familiarity with the intent of the original design team whose assumptions and design intent for the HVAC system, are typically unknown. Over time, building use may differ from original design intent in ways not foreseen by the original designers. It is not uncommon for spaces to be loaded or used in ways beyond the original design intent which may adversely impact IAQ, such as putting up walls for private offices, exceeding intended occupant densities, and bringing into the space new contaminant sources. HVAC system total capacity may be able to handle these changes from original design loads but little is done to balance the available capacity among the individual zones that may be overused or underused.

In addition, the employer may need to communicate design intent and performance criteria to building occupants whose expectations regarding their environment may exceed what is deliverable by the building systems.

To address these issues, OSHA is proposing to require that each facility have written operations and management information whose aim is twofold. One purpose is to collect, summarize and translate design assumptions and intent into operating performance criteria that impact IAQ, such as minimum outside air ventilation rates and occupant densities.

Secondly, the operations information should describe how to operate and manage the building systems so that they perform in conformance with the reported criteria. This written operations and management information replaces verbal communications and provides a training document whenever new personnel or new contractors are introduced to the site. Operating information should formally reflect changes in control strategies that typically occur in facilities to accommodate change in use or energy conservation efforts. This is an essential element because of the interdependence between outside air ventilation rate and the automatic temperature control system. In almost all buildings the performance of the ventilation system is affected by space temperature control needs.

Paragraph (c)(3)(v) proposes to require a written maintenance program for those building system components that directly affect IAQ because failure to do so may result in the degradation of IAQ in the facility. A written maintenance program is believed to be necessary because levels of HVAC system maintenance vary dramatically and sometimes are deficient where untrained personnel are designated to maintain very complex systems. The following are examples of maintenance deficiencies which have been associated with IAQ problems: plugged drains on cooling coil condensate drip pans resulting in microbial contamination of pan; failed exhaust fans in underground parking garages which allow carbon monoxide to infiltrate into the building above; microbial fouling of cooling tower water from lack of water treatment with biocides resulting in legionellosis cases; and failure of automatic temperature control system resulting in lack of outside ventilation air.

Maintenance of HVAC equipment, for example, may include simple housekeeping of equipment and air transport pathways, lube and adjustment programs for rotating machinery, and catastrophic failure maintenance to repair/replace failed equipment. There appears to be consensus among HVAC maintenance personnel that the most successful maintenance programs, gauged in terms of system performance and life-cycle economics, are proactive rather than reactive. Consequently, OSHA is promoting preventive maintenance programs for those building system components which affect IAQ. At a minimum, the maintenance program should describe the equipment to be maintained, establish maintenance procedures and frequency of performance.

Paragraph (c)(3)(vi) proposes to require a checklist to guide periodic inspections of building systems. This checklist should focus on those building system components whose failure, degradation, or misuse would adversely impact indoor air quality. The checklist shall include but not be limited to inspection of the following components and performance criteria: fibrous liner used for acoustics and insulation in airhandlers and ducts should be inspected for erosion and moisture; smoke-trails testing should be performed to verify design pressurization schemes like negative pressure smoking rooms; ceiling, floor and wall surfaces should be examined for signs of water leaks which could support and amplify microbial contamination; and outside air louvers, intake paths, dampers, actuators, and linkages should be checked for obstruction.

Paragraph (c)(5) proposes to require the establishment of records of employee complaints of building-related illnesses as part of the written program. These records are believed to be necessary to expedite review and evaluation of the system and to support implementation and operation of an adequate IAQ program [Exs. 3–434, 3–444b, 3–502].

The Agency believes that effective system operation and maintenance will necessarily rely upon written information and records such as those relating to design expectations, system capacities, code requirements, maintenance activities and system evaluations. As with other OSHA rulemakings, the written compliance plan is to be accessible to employees.

Compliance Program Implementation: Paragraph (d)

This paragraph proposes to require that the employer take certain actions to maintain acceptable indoor air quality. These actions primarily address means that OSHA believes necessary to achieve continued adequate and proper functioning of building systems [Exs. 3–10, 3–17, 3–26, 3–38, 3–41, 3–55, 3–56, 3–61, 3–85, 3–329, 3–364, 3–412, 3–415, 3–434, 3–436, 3–444A, 3–479, 3–496, 3–500, 3–501, 3–502, 3–505, 3–507, 3–529, 3–531].

Paragraph (d)(1) proposes to require that employers maintain and operate the HVAC system to provide at least the minimum outdoor air ventilation rate, based on actual occupancy, required by the applicable building code, mechanical code, or ventilation code in effect at the time the facility was

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several shortcomings that seriously reduce its effectiveness in providing incentives for firms to create safe and healthful workplaces. The scheduled benefits are significantly less than the actual losses to the injured workers, and recovery is often very difficult in the case of occupational diseases. Thus, the existence of a Workers' Compensation system limits an employer's liability significantly below the actual costs of the injury. In addition, premiums for individual firms are unlikely to be specifically related to that firm's risk environment. The firm, therefore, does not receive the proper economic signals and consequently fails to invest sufficient resources in reducing workplace injuries and illnesses. The economic costs not borne by the employer are borne by the employee or, as is often the case, by society through public insurance and welfare programs.

(4) Conclusion

OSHA believes that there are no nonregulatory alternatives that adequately protect workers from the adverse health effects associated with exposure to indoor air pollution. Tort liability laws and Workers' Compensation provide some protection, but due to market imperfections they have not been sufficient. Some employers have not complied voluntarily with standards recommended by professional organizations. The deleterious health effects resulting from continued exposure to hazardous substances require a regulatory solution.

D. Benefits

In this chapter, OSHA presents its preliminary estimates of the expected reduction in fatalities and illnesses among the employees affected by the proposed IAQ standard. A qualitative description of the non-quantifiable additional cost savings to employers, is also provided.

1. Indoor Air Quality

Health effects typically caused by poor IAQ have been categorized as Sick Building Syndrome (SBS) or Building-Related Illness (BRI). Some of the symptoms that characterize SBS include: irritation of eyes, nose and throat, dry mucous membranes and skin and coughs, hoarseness of voice and wheezing, hypersensitivity reaction, nausea and dizziness.

BRI describes specific medical conditions of known etiology such as: Respiratory allergies, legionellosis, humidifier fever, nosocomial infections, sensory irritation when caused by known agents and the symptoms and signs characteristic of exposure to chemical or biologic substances such as carbon monoxide, formaldehyde, pesticides, endotoxins or mycotoxins. BRIs do not disappear when the person leaves the building.

The Centers for Disease Control Prevention estimate that over 25,000 cases of the pneumonia caused by *Legionella* occur each year with more than 4,000 deaths. It has been suggested that a large number of these cases occur as the result of workplace exposure [Exs. 4–33, 4–318]. However, specific data on the occurrence of *Legionella*-related cases due to workplace exposure were not available.

Some of the reductions attributable to the proposed standard, such as decreases in the number of upper respiratory symptoms (nose, throat and eye symptoms) and severe headaches have been estimated. Other reductions, however, have not been quantified at this time.

OSHA's estimates are based upon the exposure profile (presented in Table VI–5) and OSHA's quantitative risk assessment discussed in detail in the preamble to the proposal). OSHA preliminarily estimates the risk of working in mechanically ventilated workplaces to be 57 severe headaches and 85 upper respiratory symptoms per 1,000 employees over a 45 year work lifetime. By applying these rates to the affected population at risk, OSHA estimates that 3.8 million severe headaches and 5.6 million upper respiratory symptoms will develop in employees over the next 45 years who work in buildings with mechanical ventilation (with the worker population held constant).

A common theme that runs through the literature and the OSHA docket indicates that the principal factor associated with indoor air quality complaints is inadequate ventilation. However, information available does not quantify the effectiveness of ventilation improvements. NEMI reports that: "ventilation system modifications and improvements are key elements of solving existing IAQ problems and reducing IAQ complaints. In every case where recommended ventilation system modifications and improvements are implemented, the frequency and severity of complaints are reduced significantly" [Ex. 3–1183].

Some of the submissions base the effectiveness of ventilation improvements on the NIOSH analysis of indoor air quality investigations [Exs. 3–1183, 3–1090]. In approximately 500 indoor air quality investigations, NIOSH found that the primary causes of indoor air quality problems were inadequate ventilation (52%), contamination from outside the building (10%), microbial contamination (5%), contamination from building fabric (4%), and unknown sources (13%). Excluding contamination from building fabric and unknown sources, this suggests that 83 percent of complaints related to IAQ problems would be eliminated by the proposed OSHA standard. For purposes of this analysis, OSHA assumes that the overall effectiveness is, therefore, 80 percent. As shown in Table VI–5, OSHA estimates that the proposed standard will prevent 3.0 million severe headaches and 4.5 million upper respiratory symptoms over the next 45 years. This is, approximately, 69,000 severe headaches and 105,000 upper respiratory symptoms per year. These estimates understate the prevalence of building-related symptoms since they only reflect excess risk in only air conditioned buildings. OSHA believes that the standard will also prevent severe headaches and upper respiratory symptoms in heated (but not air conditioned) buildings, and that it will prevent various other adverse health effects. OSHA is seeking additional information upon which to base quantifiable estimates of the other known adverse health effects.

OSHA requests comment on the methodology of estimating the benefits for the IAQ portion of the proposal. Specifically, OSHA requests any studies which document (in quantitative terms) the effectiveness of HVAC maintenance on the decline of indoor air related ailments.

2. Environmental Tobacco Smoke

Tobacco smoke has been classified as a carcinogen by the International Agency for Research on Cancer, the Surgeon General, NIOSH, and the U.S. Environmental Protection Agency. The National Health Interview Survey of Cancer Epidemiology and Control (NHIS-CEC) shows that the prevalence of cigarette smoking continues to decline in smoking among adults by approximately 0.50 percent per year. Despite these declines, smoking is responsible for an estimated 390,000 deaths. Exposure to ETS has been associated with the occurrence of many diseases, such as lung cancer and heart disease in nonsmokers and low birthweight in the offspring of nonsmokers.

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smoking of tobacco products is permitted.

Director means the Director, National Institute for Occupational Safety and Health (NIOSH) U.S. Department of Health and Human Services or designee.

Employer means all persons defined as employers by Sec. 3(5) of the Occupational Safety and Health Act of 1970 including employers (such as building owners or lessees) who control the ventilation or maintenance of premises where employees of other employers work.

HVAC system means the collective components of the heating, ventilation and air-conditioning system including, but not limited to, filters and frames, cooling coil condensate drip pans and drainage piping, outside air dampers and actuators, humidifiers, air distribution ductwork, automatic temperature controls, and cooling towers.

Nonindustrial work environment means an indoor or enclosed work space such as, but not limited to, offices, educational facilities, commercial establishments, and healthcare facilities, and office areas, cafeterias, and break rooms located in manufacturing or production facilities used by employees. Non-industrial work environments do not include manufacturing and production facilities, residences, vehicles, and agricultural operations.

Renovation and remodeling means building modification involving activities that include but are not limited to: removal or replacement of walls, ceilings, floors, carpet, and components such as moldings, cabinets, doors, and windows; painting, decorating, demolition, surface refinishing, and removal or cleaning of ventilation ducts.

(c) *Indoor air quality (IAQ) compliance program.*

(1) All employers with workplaces covered by paragraph (a)(1) of this section shall establish a written IAQ compliance program.

(2) The employer shall identify a designated person who is given the responsibility to assure implementation of the IAQ compliance program.

(3) Written plans for compliance programs shall include at least the following:

(i) A written narrative description of the facility building systems;

(ii) Single-line schematics or as-built construction documents which locate major building system equipment and the areas that they serve;

(iii) Information for the daily operation and management of the building systems, which shall include at least a description of normal operating procedures, special procedures such as seasonal start-ups and shutdowns, and a list of operating performance criteria including, but not limited to minimum outside air ventilation rates, potable hot water storage and delivery temperatures, range of space relative humidities, and any space pressurization requirements;

(iv) A general description of the building and its function including but not limited to, work activity, number of employees and visitors, hours of operation, weekend use, tenant requirements and known air contaminants released in the space;

(v) A written maintenance program for the maintenance of building systems which shall be preventive in scope and reflect equipment manufacturer's recommendations and recommended-good-practice as determined by the building systems maintenance industry. At a minimum, the maintenance program shall describe the equipment to be maintained, and establish maintenance procedures and frequency of performance;

(vi) A checklist for the visual inspection of building systems.

(4) The following additional information, if available, shall be retained by the employer to assist in potential indoor air quality evaluations:

(i) As-built construction documents;

(ii) HVAC system commissioning reports;

(iii) HVAC systems testing, adjusting and balancing reports;

(iv) Operations and maintenance manuals;

(v) Water treatment logs; and

(vi) Operator training materials.

(5) The employer shall establish a written record of employee complaints of signs or symptoms that may be related to building-related illness to include at least information on the nature of the illness reported, number of employees affected, date of employee complaint, and remedial action, if any, taken to correct the source of the problem.

(d) *Compliance program implementation.* Employers shall assure compliance with this section by implementing at least the following actions:

(1) Maintain and operate the HVAC system to assure that it operates up to original design specifications and continues to provide at least the minimum outside air ventilation rate, based on actual occupancy, required by the building code, mechanical code, or ventilation code applicable at the time the facility was constructed, renovated, or remodeled, whichever is most recent;

(2) Conduct building systems inspections and maintenance in accordance with paragraph (c) of this section;

(3) Assure that the HVAC system is operating during all work shifts, exce during emergency HVAC repairs and during scheduled HVAC maintenance;

(4) Implement the use of general or local exhaust ventilation where housekeeping and maintenance activities involve use of equipment or products that could reasonably be expected to result in hazardous chemical or particulate exposures to employees working in other areas of the building or facility;

(5) Maintain relative humidity belo 60% in buildings with mechanical cooling systems;

(6) The employer shall monitor carbon dioxide levels when routine maintenance under paragraph (d)(1) c this section is done. When the carbon dioxide level exceeds 800 ppm, the employer shall check to make sure the HVAC system is operating as it shoul If it is not, the employer shall take necessary steps to correct deficiencies if they exist.

(7) Assure that buildings without mechanical ventilation are maintaine so that windows, doors, vents, stacks and other portals designed or used for natural ventilation are in operable condition;

(8) Assure that mechanical equipmen rooms and any non-ducted air plenums or chases that transport air are maintained in a clean condition, hazardous substances are properly stored to prevent spillage, and asbestos, if friable, is encapsulated or removed that it does not enter the air distribut system;

(9) Assure that inspections and maintenance of building systems are performed by or under the supervisic of the designated person;

(10) Establish a written record of building system inspections and maintenance required to be performe under this section;

(11) Assure that employees performing work on building systems are provided with and use appropria personal protective equipment as prescribed in 29 CFR part 1926, subpart E, Personal Protective and Life Saving Equipment; 29 CFR part 1926.52, Occupational Noise Exposure; 29 CF part 1910, subpart I, Personal Protective Equipment; and 29 CFR part 1910.95, Occupational Noise Exposure;

(12) Evaluate the need to perform alterations of the building systems to meet the minimum requirements specified in paragraph (d) of this sect in response to employee complaints building-related illnesses; and

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maintenance chemicals and pesticides [Exs. 3–56, 3–436, 3–496, 3–500, 3–505].

With respect to tobacco smoke in workplaces where smoking is not prohibited, paragraph (e)(1) proposes to require the establishment of designated smoking areas. Such areas must be enclosed and exhausted directly to the outside, and maintained under negative pressure sufficient to contain tobacco smoke within the designated area. Smoking is not permitted during cleaning and maintenance work in these designated smoking areas. Moreover, although cleaning and maintenance are specified in this paragraph, it is OSHA's intent that no work of any kind shall be performed in a designated smoking area when smoking is taking place. Designated smoking areas must be areas where employees do not have to enter in the performance of normal work activities. Signs must also be posted at designated smoking areas. Signs must be posted to inform anyone entering the building that smoking is restricted to designated areas. Finally, smoking within designated areas is not permitted during any time that the exhaust ventilation system servicing that area is not operating properly.

The proposed provisions under paragraph (e)(1) addressing control of tobacco smoke are intended to ensure that employees outside of the designated smoking area will not be exposed to ETS. The Agency anticipates that the provisions as proposed will accomplish that goal. Enclosing smoking areas, exhausting them to the outside, maintaining them under negative pressure, and prohibiting smoking in designated areas even when the exhaust system is inoperable are believed to be necessary and sufficient to prevent tobacco smoke from migrating to other areas of the building.

The designated smoking area must be under negative pressure compared to all surrounding spaces including adjoining rooms, corridors, plenums and chases. Negative pressure is achieved by exhausting more air from the space than is supplied to the space.

Transfer air must enter the designated smoking room to make-up the volumetric flowrate differential between supply and exhaust air. It may be necessary to provide a tight architectural enclosure so as to achieve negative pressure and containment. Leakage through a lay-in ceiling tile system may occur if there is a return air plenum above it. Negative pressure will induce airflow into the room through the entrance door undercut. Containment may be checked by using smoke-trails at the door undercut to verify direction of airflow.

Contaminated exhaust air from a designated smoking room must be transported to the outside through exhaust ducts under negative pressure to avoid duct leakage into nonsmoking areas that the duct passes through.

The provisions regarding posting of signs are intended to prevent inadvertent entry into smoking areas, and inadvertent smoking in areas other than designated smoking areas. To prevent involuntary exposure, designated smoking areas cannot be areas where employees perform normal work activities. For the same reason, smoking is not permitted in smoking areas during performance of work activities such as cleaning and maintenance of the designated smoking area.

This provision will have special impact on establishments such as bars and restaurants. OSHA invites comments on feasibility considerations relative to such establishments and suggestions for alternative ways to assure that nonsmoking workers will not be exposed to tobacco smoke there.

Proposed paragraph (e)(2) establishes requirements dealing with outdoor air pollutants and contaminants emitted locally within workspaces. This paragraph proposes to require the employer to implement measures to restrict the entry of outdoor air pollutants into the building and to control local indoor sources of air contaminant emissions by employing other control measures like substitution or local source capture exhaust ventilation.

Proposed paragraph (e)(3) proposes to require the control of microbial contamination by routinely inspecting for and repairing water leaks that can promote growth of biologic agents, by promptly drying, replacing, removing, or cleaning damp or wet materials; and by taking measures to remove visible microbial contamination in ductwork, humidifiers, other HVAC system components, or on other building surfaces.

Proposed paragraph (e)(4) addresses the use of cleaning and maintenance chemicals, pesticides and other hazardous chemicals. Pesticides must be used according to manufacturers' recommendations, and where chemicals are to be used, employees in those areas affected are to be informed, at least within 24 hours prior to use, of the type of chemical to be applied.

The provisions proposed under (e)(2) are intended to ensure that indoor air quality is not degraded as a result of entry of outdoor contaminants, such as vehicle exhaust, or by circulation of contaminants generated within the building. The Agency believes that, where necessary, entry of outdoor air pollutants can be restricted by eliminating or repositioning entry points into the building.

Indoor local contaminant emissions can be minimized where necessary, through application of control measures such as source substitution and engineering controls that may include local source capture exhaust ventilation. Collection of contaminants at their source of emission through engineering controls is an accepted basic principle of industrial hygiene. Equipment and processes which are located or take place in areas that may lead to contamination of other areas should be provided with engineering controls, where necessary and feasible.

The provisions proposed in paragraph (e)(3) are intended to limit the opportunity for microbiological contamination of building systems and structures. Although individual microbes are not visible to the naked eye, colonies of microbes are. Moisture can lead to microbiological growth in indoor spaces, within HVAC systems, or within building structures, and thus to a variety of detrimental health effects. The employer therefore, is required to take preventive and corrective actions to minimize microbiological growth. Preventive action includes routine inspection for biological growth, with required corrective actions such as repairing water leaks, drying, replacing, or cleaning wet materials, and removal of visible microbiological growth (Exs. 3–61, 3–502).

The provisions proposed in paragraph (e)(4) are intended to restrict indoor exposure to hazardous substances such as pesticides and chemicals used for cleaning and maintenance purposes. The Agency believes that proper use of such substances is important to limit incidental exposures to those performing cleaning and maintenance as well as to other employees who might be incidentally exposed. Manufacturers recommendations for use of these products often address aspects of ventilation, employee protection, occupancy limitations, and other protective measures. Thus, the Agency has proposed to require that chemicals covered under this paragraph must be used in accordance with manufacturer's recommendations. To further limit the potential for incidental exposures to these chemicals the standard proposes to require that employees in areas to be treated by such chemicals are to be notified within at least 24 hours prior to their application.

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asymptomatic arterial wall injury may occur which can contribute to cardiac injury in the future. However, an older person with pre-existing coronary artery disease and therefore minimum cardiac reserve may not be able to tolerate short-term ETS exposure, due to the increased stress on the heart.

5. Reproductive Effects

Data on the reproductive effects due to the exposure of nonsmoking pregnant women to ETS has been presented in many studies [Exs. 3–438, 4–92, 4–132, 4–174, 4–208, 4–273, 4–285, 4–287, 4–299]. This is important since many nonsmoking women continue to work throughout their pregnancies. Pregnant women working in indoor environments without tobacco smoking restrictions, as in restaurants, comprise one of the most heavily ETS-exposed groups [Exs. 4–151, 4–287].

Low birthweight has also been shown to be associated with paternal smoking, implying passive exposure to tobacco smoke by the nonsmoking mother [Exs. 4–92, 4–273]. Passive exposure to tobacco smoke is estimated to double the risk of low birthweight in a full-term baby [Ex. 4–208]. Nonsmoking pregnant women who are exposed to ETS have been reported to deliver neonates that range 24 to 120 grams lighter in weight than those babies delivered by nonexposed pregnant women [Exs. 4–132, 4–174, 4–208, 4–273]. This relationship between passive smoking and low birthweight remains statistically significant even after accounting for mother's age, parity, social class, sex of baby, and alcohol consumption. This effect is more apparent in neonates born to actively smoking women who deliver babies that weigh, on average, 200 grams less than those of nonsmoking women [Ex. 4–101]. The reduction in birthweight is clinically significant at the low end of the birthweight distribution. These infants have higher perinatal mortality [Ex. 4–239].

Other reproductive effects that have been ascribed to maternal ETS exposure include miscarriage, an increase in congenital abnormalities [Exs. 4–239, 4–299], and numerous other physiological effects [Ex. 4–297]. It was reported that these effects may be part of a general immunosuppressive condition associated with the occurrence of low birthweight [Ex. 4–299]. This effect may predispose the baby to respiratory tract infections.

The effects of environmental smoke exposure on the fetus may have long-term sequelae into childhood and adulthood [Exs. 4–53, 4–181, 4–213, 4–225, 4–239, 4–51, 4–297]. There is limited evidence which suggests that growth retardation observed in the fetus is reflected in the growing child as reductions in lung development [3–438]. This is especially relevant if that child continues to be exposed to ETS throughout childhood and into adulthood [Exs. 4–177, 4–297]. Prenatal exposure to ETS and exposure to ETS as a child may also increase an individual's cancer risk, perhaps by a factor of two (2) [Exs. 4–65, 4–164, 4–252].

Experimental research on the adverse reproductive effects associated with ETS exposure in animals is limited. However, one study [Ex. 4–6] demonstrated such effects. Sciatic nerve tissue taken from the offspring of ETS-exposed female mice revealed definite toxic effects on the neonatal tissue [Ex. 4–6]. Pregnant female mice (C57BL/Ks]) were exposed to low-tar cigarette smoke in a special smoking chamber. Cigarette smoke was blown into the chamber for 4 minutes, 5 times daily, except on weekends when this was done 3 times daily. At 18 days of gestation, blood samples were taken and carbon monoxide levels were measured. Ultrastructural abnormalities of fetal tissue revealed swollen mitochondria with distorted cristae, some indication of deformed mitochondria, darkened nuclei with condensations of nuclear material, lamellar bodies, granules and myelin bodies similar to those found in human toxicity studies. The blood samples from pregnant mice revealed a mean carbon monoxide saturation in the hemoglobin of 9% which is equivalent to that found in humans who actively smoke 10–20 cigarettes per day.

6. Cancer

Concern over the carcinogenic effects of ETS was expressed in many comments submitted to the docket, such as Exs. 3–32, 3–35, 3–38, 3–207, 3–438, 3–440A, and 3–449. The results of epidemiological and experimental studies indicate that exposure to ETS is causally associated with cancer of the lung in chronically-exposed nonsmokers. A discussion of this evidence follows.

(a) Evidence of Association.—The results of epidemiological studies taken in the aggregate suggest that nonsmoker exposure to ETS is causally-related to the development of lung cancer.

Evidence of specificity of effect is provided by active smoking studies that report a causal association with lung cancer [Ex. 4–311]. It was therefore logical to examine nonsmokers with passive exposure to tobacco smoke, since the chemicals found in passive smoke are qualitatively similar to those in mainstream smoke. Active smoking induces all four major histological types of human lung cancer—squamous-cell carcinomas, small-cell carcinomas, large-cell carcinomas, and adenocarcinomas [Ex. 4–311]. The results of lung cancer studies that examined the variation in tumor cell type induced by ETS exposure indicate that mostly adenocarcinomas and squamous cell carcinomas are produced by ETS exposure. Some studies have reported an excess of adenocarcinomas, while others have reported excesses in squamous cell and small-cell carcinomas. From this information, it is apparent that similar tumor cell types are induced by ETS exposure as are induced by active smoking.

The unequivocal causal association between active tobacco smoking and lung cancer in humans, as well as the corroborative evidence of the carcinogenicity of tobacco smoke provided by animal bioassays and *in vitro* studies and the chemical similarity between mainstream smoke and ETS, clearly establish the plausibility that ETS is also a human lung carcinogen (Table II–2). In addition, biomarker studies verify that ETS exposure results in detectable uptake of tobacco constituents by nonsmokers [Exs. 4–50, 4–311].

TABLE II–2.—43 CHEMICAL COMPOUNDS IDENTIFIED IN TOBACCO SMOKE FOR WHICH THERE IS "SUFFICIENT EVIDENCE" OF CARCINOGENICITY IN HUMANS OR ANIMALS [EX. 4–160]

Acetaldehyde
Acylonitrile
Arsenic
Benz (a)anthracene
Benzene
Benzo (a)pyrene
Benzo(b)fluoranthene
Benzo (k)fluoranthene
Cadmium
Chromium VI
DDT
Dibenz(a,h)acridine
Dibenz(a,j)acridine
Dibenz(a,h)anthracene
Dibenzo (a,i)pyrene
Dibenzo (a,e)pyrene
Dibenzo (a,l)pyrene
Dibenzo (a,h)pyrene
Formaldehyde
Hydrazine
Lead
Nickel
N-nitrosodiethanolamine
N-nitrosodiethylamine
N'-nitrosodimethylamine
N'-nitrosonornicotine
N-nitrosopiperidine
N-nitrosodi-n-propylamine
N-nitrosopyrrolidine
N-nitrosodi-n-butylamine
ortho-toluidine

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information and additional data, views and arguments from any person who has participated in the oral proceedings.

OSHA recognizes that there may be interested persons or organizations who, through their knowledge of the subject matter or their experience in the field, would wish to endorse or support the whole proposal or certain provisions of the proposal. OSHA welcomes such supportive comments, including any pertinent data and cost information which may be available, in order that the record of this rulemaking will present a balanced picture of the public response on the issues involved.

XII. List of Subjects

29 CFR Parts 1910, 1915 and 1926

Hazardous substances, Indoor air quality, Occupational Safety and Health, Reporting and recordkeeping requirements.

29 CFR Part 1928

Occupational Safety and Health.

XIII. Authority and Signature

This document was prepared under the direction of Joseph A. Dear, Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, 200 Constitution Avenue NW., Washington, DC 20210. Pursuant to sections 6(b) and 8(c) and 6(g)(2) of the Act, OSHA hereby proposes to amend 29 CFR by adding a new § 1910.1033, 1915.1033, 1926.1133 and revising of § 1910.19 and 1928.21 as set forth below.

Signed at Washington, DC, this 28th day of March, 1994.

Joseph A. Dear,

Assistant Secretary for Occupational Safety and Health.

Part 1910, 1915, 1926, and 1928 of title 29 of the Code of Federal Regulations (CFR) are hereby proposed to be amended as follows:

XIV Standards

Part 1910, 1915, 1926 [AMENDED]—OCCUPATIONAL SAFETY AND HEALTH STANDARDS

1. The authority citation for subpart B of part 1910 would continue to read as follows:

Authority: Secs. 4, 6, and 8 of the Occupational Safety and Health Act, 29 U.S.C. 653, 655, 657; Walsh-Healey Act, 41 U.S.C. 35 *et seq.*; Service Contract Act of 1965, 41 U.S.C. 351 *et seq.*, sec. 107, Contract Work Hours and Safety Standards Act (Construction Safety Act), 40 U.S.C. 333; sec. 41, Longshore and Harbor Workers' Compensation Act, 33 U.S.C. 942; National Foundation of Arts and Humanities Act, 20 U.S.C. 951 *et seq.*; Secretary of Labor's Order No. 12–71 (36 FR 8754), 8–76 (41 FR 1911), 9–83 (48 FR 35736), or 1–90 (55 FR 9033), as applicable.

2. The authority citation for subpart Z of Part 1910 would continue to read as follows:

Authority: Secs. 6, 8 of the Occupational Safety and Health Act, 29 U.S.C. 653, 655, 657; Secretary of Labor's Order No. 12–71 (36 FR 8754), 8–76 (41 FR 1911), 9–83 (48 FR 35736), or 1–90 (55 FR 9033), as applicable; and 29 CFR part 1911.

All of subpart Z issued under section 6(b) of the Occupational Safety and Health Act, except those substances which have exposure limits listed in Tables Z–1, Z–2, and Z–3 of 29 CFR 1910.1000. The latter were issued under Section 6(a) (29 U.S.C. 655(a)).

Section 1910.1000, Tables Z–1, Z–2, and Z–3 also issued under 5 U.S.C. 533. Section 1910.1000, Tables Z-1, Z-2, and Z-3 were not issued under 29 CFR part 1911 except for the arsenic (organic compounds), benzene and cotton dust listings.

Section 1910.1001 also issued under Sec. 107 of Contract Work Hours and Safety Standards Act, 40 U.S.C. 333.

Section 1910.1002 not issued under 29 U.S.C. 655 or 29 CFR part 1911; also issued under 5 U.S.C. 553.

Section 1910.1025 also issued under 5 U.S.C. 553.

Section 1910.1043 also issued under 5 U.S.C. 551 *et seq.*

Sections 1910.1200, 1910.1499, and 1910.1500 also issued under 5 U.S.C. 553.

3. The authority citation for part 1915 would continue to read as follows:

Authority: Sec. 41, Longshore and Harbor Workers Compensation Act (33 U.S.C. 941); secs. 4, 6, 8 Occupational Safety and Health Act of 1970 (29 U.S.C. 653, 655, 657); sec. 4 of the Administrative Procedure Act (5 U.S.C. 553); Secretary of Labor's Order No. 12–71 (36 FR 8754), 8–76 (41 FR 25059), 9–83 (48 FR 35736) or 1–90 (55 FR 9033), as applicable; 29 CFR part 1911.

4. The authority citation for subpart Z of part 1926 would be revised to read as follows:

Authority: Sec. 107, Contract Work Hours and Safety Standards Act (Construction Safety Act) (40 U.S.C. 333); Secs. 6, 8 of the Occupational Safety and Health Act, 29 U.S.C. 653, 655, 657; Secretary of Labor's Order No. 12–71 (36 FR 8754), 8–76 (41 FR 1911), 9–83 (48 FR 35736), or 1–90 (55 FR 9033), as applicable; and 29 CFR part 1911.

Section 1926.1102 not issued under 29 U.S.C. 655 or 29 CFR part 1911; also issued under 5 U.S.C. 653.

Section 1926.1103 through 1926.1118 also issued under 29 U.S.C. 653.

Section 1926.1128 also issued under 29 U.S.C. 653.

Section 1926.1145 and 1926.1147 also issued under 29 U.S.C. 653.

Section 1926.1148 also issued under 29 U.S.C. 653.

5. Section 1910.19 of subpart B of part 1910 is proposed to be amended by adding a paragraph (l) to read as follows:

§ 1910.19 Special provisions for air contaminants

* * * * *

(l) *Indoor air quality.* Section 1910.1033 shall apply to the exposure of every employee in every employment covered by § 1910.16.

6. Subpart Z of parts 1910, 1915, 1926 of Title 29 of the Code of Federal Regulations is proposed to be amended by adding identical new sections as 1910.1033, 1915.1033 and 1926.1133 to read as follows:

§ _____.____ Indoor air quality.

(a) *Scope and application.* (1) The provisions set forth in this section apply to all nonindustrial work environments.

(2) The provisions set forth in paragraph (e)(1) of this section, which address employee exposure to tobacco smoke, apply to all indoor or enclosed workplaces under OSHA jurisdiction.

(b) *Definitions.*

Air contaminants refers to substances contained in the vapors from paint, cleaning chemicals, pesticides, and solvents, particulates, outdoor air pollutants and other airborne substances which together may cause material impairment to employees working within the nonindustrial environment.

Assistant Secretary means the Assistant Secretary of Labor for Occupational Safety and Health, U.S. Department of Labor, or designee.

Building-related illness describes specific medical conditions of known etiology which can be documented by physical signs and laboratory findings. Such illnesses include sensory irritation when caused by known agents, respiratory allergies, asthma, nosocomial infections, humidifier fever, hypersensitivity pneumonitis, Legionnaires' disease, and the signs and symptoms characteristic of exposure to chemical or biologic substances such as carbon monoxide, formaldehyde, pesticides, endotoxins, or mycotoxins.

Building systems include but are not limited to the heating, ventilation and air-conditioning (HVAC) system, the potable water systems, the energy management system and all other systems in a facility which may impact indoor air quality.

Designated person means a person who has been given the responsibility by the employer to take necessary measures to assure compliance with this section and who is knowledgeable in the requirements of this standard and the specific building systems servicing the affected building or office.

Designated smoking area means a room, in a non-work area, in which

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likely to have different deposition and exposure patterns for the various cigarette smoke components in the respiratory system. For example, rodents have extensive and complex nasal turbinates where significant particle deposition could occur, decreasing exposure to the lung. These anatomical and physiological differences, aside from the subchronic exposure, may partially account for absence of any lung tumors in the study by Coggins et al.

The application of cigarette smoke condensate (CSC) to mouse skin is a widely employed assay for the evaluation of carcinogenic potential. CSC assays may not, however, reveal all of the carcinogenic activity of actual cigarette smoke, because these condensates lack most of the volatile and semi-volatile components of whole smoke. Benign skin tumors and carcinomas were seen in Swiss-ICR mice exposed to cigarette tar from the sidestream smoke of nonfilter cigarettes suspended in acetone and applied to skin for 15 months [Ex. 4–327]. In lifetime rat studies, intrapulmonary implants of mainstream smoke condensate in a lipid vehicle caused a dose-dependent increase in the incidence of lung carcinomas [Exs. 4–75, 4–289].

The polyamines contained in tobacco smoke, spermidine, spermine, and their diamine precursor, putrescine, are believed to have an essential role in cellular proliferation and differentiation. Formation of putrescine from ornithine is catalyzed by ornithine decarboxylase (ODC), the rate-limiting enzyme in polyamine biosynthesis. A significant increase in lung and trachea ornithine decarboxylase activity was observed by Olsen [Ex. 4–245] after an eight week exposure of male Sprague-Dawley rats to MS smoke. All dilutions of SS smoke exposure caused significant increase in trachea ODC activity but did not influence the lung ODC activity.

Environmental tobacco smoke induced carcinogenicity is also supported by a case-control study of lung cancer in pet dogs [Ex. 4–259]. The study compared the incidence of lung cancer in pet dogs exposed to their owners' smoking versus dogs whose owners did not smoke. Dogs have a very low natural incidence of lung cancer. There was an elevated risk of lung cancer (Relative Risk = 1.6) observed in pets with smoking owners. However, the analysis was statistically insignificant, perhaps in part due to small sample size.

7. Genotoxicity

Short-term mutagenicity tests have gained widespread acceptance as an initial step in the identification of potential carcinogens. Extensive use of these tests has come about because they are easy to perform and are inexpensive and also because of the reported high positive correlations between short-term mutagenicity tests and carcinogenicity. It has been reported that 90 percent of the carcinogens tested are mutagens and 90 percent of the noncarcinogens are nonmutagens.

Several short-term bioassays have been performed to evaluate the genotoxicity of cigarette smoke. While most of them have evaluated the effect of cigarette smoke condensate, some have attempted to evaluate either the gas phase or the whole smoke.

The most commonly employed assay for mutagenic activity employs various strains of *Salmonella typhimurium*. Whole smoke as well as cigarette smoke condensate of tobacco have been shown to be mutagenic in *Salmonella typhimurium* strain TA 1538 [Ex. 4–21]. Sidestream smoke was also found to be mutagenic in a system where the smoke was tested directly on the bacterial plates [Ex. 4–246]. Sidestream smoke and extracts of ETS collected from indoor air [Exs. 4–202, 4–5, 4–198, 4–201, 4–203] also exhibited mutagenic activity in this bacterial strain. Claxton et al. [Ex. 4–55] found that sidestream smoke accounted for approximately 60% of the total *S. typhimurium* mutagenicity per cigarette, 40% from the sidestream smoke particulates and 20% from the semi-volatiles. The highly volatile fraction, from either mainstream or sidestream smoke was not mutagenic.

Condensates from both mainstream [Exs. 4–89, 4–193] and sidestream smoke [Ex. 4–90] have also been reported to have mutagenic activity. Doolittle et al. [Ex. 4–89] demonstrated the genotoxicity of the sidestream smoke from the Kentucky Reference cigarette (1R4F) by employing several different assays. In their study, sidestream smoke produced positive results in *Salmonella typhimurium* strains TA98, TA100, TA1537, and TA1538 in the presence of S9 mix from aroclor-induced rat liver but produced negative results in strain TA1535. They also showed that sidestream smoke produced positive results in the Chinese hamster ovary cells chromosomal aberration assay and in the Chinese hamster ovary cell sister-chromatid exchange assay both with and without metabolic activation. They demonstrated that the sidestream smoke was weakly positive in inducing DNA repair in cultured rat hepatocytes. However, sidestream smoke was nonmutagenic in the Chinese hamster ovary cell-HGPRT assay both with and without metabolic activation but it was found to be cytotoxic in this system.

In their further studies, Doolittle et al. [Ex. 4–90] observed similar responses when they measured the genotoxic activity of mainstream cigarette smoke condensate (CSC) from Kentucky reference research cigarette (1R4F). As seen with sidestream smoke, CSC in this study was mutagenic in *Salmonella typhimurium* strain TA98, TA100, TA1537, and TA1538 in the presence of S9 mix but was negative in strain TA1535. CSC was also positive in the Chinese hamster ovary (CHO) cells-chromosomal aberration assay and in the CHO-sister-chromatid exchange assay both with and without metabolic activation. CSC was weakly positive in inducing DNA repair in cultured rat hepatocytes. However, again as seen with sidestream smoke, CSC was nonmutagenic in the CHO–HGPRT assay, with or without metabolic activation but was found to be cytotoxic in this system. The results from these two studies appear to indicate that sidestream smoke behaves very much like mainstream smoke in these assays.

Mohtashamipur et al. [Ex. 4–227] demonstrated significant mutagenic activity in the urine of rats exposed to sidestream smoke. In this study, cigarettes were machine smoked under standardized laboratory conditions and the sidestream smoke of two cigarettes was directed through metabolism cages containing rats. The urine of these rats was collected 24 hours prior to the SS exposure and 24 hours after the onset of the exposure. The individual urine samples of all (10) rats after exposure showed significantly higher activity for direct-acting mutagens (in strain TA1538) than the urine samples of the same rats before the exposure.

The formation of DNA adducts is widely accepted as an initial step in the carcinogenesis process. The measurement of DNA adducts by the ^{32}P-postlabeling assay has been used as a way to assess DNA damage following exposure to cigarette smoke. Lee et al. [Ex. 4–194] exposed Sprague-Dawley rats to 0.1, 1.0 and 10 mg total particulate matter/m^3 of aged and diluted sidestream smoke (ADSS) for 6 hours per day for 14 consecutive days. They examined the DNA from lung, heart, larynx and liver after 7 and 14 days of exposure and after 14 days of recovery. They also examined alveolar macrophages for chromosomal aberrations. Exposure related DNA adducts were found in the highest dose

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Public reporting burden for this collection of information is estimated to average five minutes per response. Send any comments regarding this burden estimate, or any other aspect of this collection of information, including suggestions for reducing this burden, to the Office of Information Management, Department of Labor, room N-1301, 200 Constitution Avenue, NW., Washington, DC 20210; and to the Office of Information and Regulatory Affairs, Office of Management and Budget, Washington, DC 20503.

XI. Public Participation

Interested persons are requested to submit written data, views and arguments concerning this proposal. Responses to the questions raised at various places in the proposal are particularly encouraged. These comments must be postmarked by June 29, 1993. Comments are to be submitted in quadruplicate or 1 original (hardcopy) and 1 disk (5¼ or 3½) in WP 5.0, 5.1, 6.0 or Ascii. Note: Any information not contained on disk, e.g., studies, articles, etc., must be submitted in quadruplicate to: The Docket Office, Docket No. H-122, room N-2625, U.S. Department of Labor, 200 Constitution Avenue, NW., Washington, DC 20210, Telephone No. (202) 219-7894.

All written comments received within the specified comment period will be made a part of the record and will be available for public inspection and copying at the above Docket Office address.

Notice of Intention To Appear at the Informal Hearing

Pursuant to section 6(b)(3) of the OSH Act, informal public hearings will be held on this proposal in Washington, DC from July 12 through July 26, 1994. If OSHA receives sufficient requests to participate in the hearing, the hearing period may be extended.

The hearing will commence at 9:30 a.m. in the auditorium of the Frances Perkins Building, U.S. Department of Labor, 3rd Street and Constitution Avenue NW., Washington, DC 20210.

Persons desiring to participate at the informal public hearing must file a notice of intention to appear by June 20, 1994. The notice of intention to appear must contain the following information:

1. The name, address, and telephone number of each person to appear;

2. The capacity in which the person will appear;

3. The approximate amount of time required for the presentation;

4. The issues that will be addressed;

5. A brief statement of the position that will be taken with respect to each issue; and

6. Whether the party intends to submit documentary evidence and, if so, a brief summary of it.

The notice of intention to appear shall be mailed to Mr. Thomas Hall, OSHA Division of Consumer Affairs, Docket No. H-122, U.S. Department of Labor, room N-3647, 200 Constitution Avenue, NW., Washington, DC 20210, telephone (202) 219-8615.

A notice of intention to appear also may be transmitted by facsimile to (202) 219-5986, by the same date provided the original and 3 copies are sent to the same address and postmarked no later than 3 days later.

Filing of Testimony and Evidence Before the Hearing

Any party requesting more than ten (10) minutes for presentation at the informal public hearing, or who intends to submit documentary evidence, must provide in quadruplicate the testimony and evidence to be presented at the informal public hearing. One copy shall not be stapled or bound and be suitable for copying. These materials must be provided to Mr. Thomas Hall, OSHA Division of Consumer Affairs at the address above and be postmarked no later than June 29, 1994.

Each submission will be reviewed in light of the amount of time requested in the notice of intention to appear. In instances where the information contained in the submission does not justify the amount of time requested, a more appropriate amount of time will be allocated and the participant will be notified of that fact prior to the informal public hearing.

Any party who has not substantially complied with the above requirement may be limited to a ten-minute presentation and may be requested to return for questioning at a later time.

Any party who has not filed a notice of intention to appear may be allowed to testify for no more than 10 minutes as time permits, at the discretion of the Administrative Law Judge, but will not be allowed to question witnesses.

Notice of intention to appear, testimony and evidence will be available for inspection and copying at the Docket Office at the address above.

Conduct and Nature of Hearing

The hearing will commence at 9:30 a.m. on the first day. At that time, any procedural matters relating to the proceeding will be resolved.

The nature of an informal rulemaking hearing is established in the legislative history of section 6 of the OSH Act and is reflected by OSHA's rules of procedure for hearings (29 CFR 1911.15(a)). Although the presiding officer is an Administrative Law Judge and questioning by interested persons is allowed on crucial issues, the proceeding is informal and legislative in type. The Agency's intent, in essence, is to provide interested persons with an opportunity to make effective oral presentations which can proceed expeditiously in the absence of procedural restraints which impede or protract the rulemaking process.

Additionally, since the hearing is primarily for information gathering and clarification, it is an informal administrative proceeding rather than an adjudicative one. The technical rules of evidence, for example do not apply. The regulations that govern hearings and the pre-hearing guidelines to be issued for this hearing will ensure fairness and due process and also facilitate the development of a clear, accurate and complete record. Those rules and guidelines will be interpreted in a manner that furthers that development. Thus, questions of relevance, procedure and participation generally will be decided so as to favor development of the record.

The hearing will be conducted in accordance with 29 CFR part 1911. It should be noted that § 1911.4 specifies the Assistant Secretary may upon reasonable notice issue alternatives procedures to expedite proceedings or for other good cause. The hearing will be presided over by an Administrative Law Judge who makes no decision or recommendation on the merits of OSHA's proposal. The responsibility of the Administrative Law Judge is to ensure that the hearing proceeds at a reasonable pace and in an orderly manner. The Administrative Law Judge, therefore, will have all the powers necessary and appropriate to conduct a full and fair informal hearing as provided in 29 CFR part 1911 including the powers:

1. To regulate the course of the proceedings;

2. To dispose of procedural requests, objections and comparable matters;

3. To confine the presentations to the matters pertinent to the issues raised;

4. To regulate the conduct of those present at the hearing by appropriate means;

5. In the Judge's discretion, to question and permit the questioning of any witness and to limit the time for questioning; and

6. In the Judge's discretion, to keep the record open for a reasonable, stated time (known as the post-hearing comment period) to receive written

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(3) Are the proposed provisions addressing control of microbial contamination effective, feasible, or necessary? Why? Why not? What additional provisions, if any, should be included to preclude microbial contamination for adversely affecting indoor air quality?

(4) Where hazardous chemicals are used in the workplace, including cleaning and maintenance chemicals, is employee notification of their use 24 hours prior to their application, as proposed, necessary to mitigate potential incidental exposure to such chemicals? To what extent does the use of such chemicals in nonindustrial environments present a health risk to other employees, or to the acceptability of indoor air quality? Which chemicals and their uses are of particular concern in non-industrial indoor environments?

(5) Are the proposed provisions specifically addressing renovation and remodeling activities necessary and appropriate and why? Particularly, are the proposed requirements to develop a work plan focusing special attention on HVAC systems, area isolation or containment, and air contaminant suppression controls necessary to limit the potential for degradation of air quality? Why? Why not? What other provisions, if any, should be included to limit the effects that renovation and remodeling activities may have on indoor environments?

Employee Information and Training, Paragraph (g)

(1) Are the provisions proposing that building systems maintenance workers receive special training with respect to the use of personal protective equipment, use of ventilation during cleaning and maintenance activities, and on proper use and disposal of hazardous chemicals and other agents, necessary and appropriate to assure maintenance of acceptable indoor air quality? Why? Why not?

(2) Should training of building maintenance and systems workers include additional specific elements not proposed in this notice? What should this additional training consist of and why? Which workers should this training be provided to—all maintenance and building systems workers, supervisors, crew leaders? Should such training be provided periodically, or would initial training suffice?

(3) Is it necessary, as proposed, to require that all employees in the facility be informed of the contents of the standard and of signs and symptoms associated with building-related illness? Why? Why not?

Recordkeeping, Paragraph (h)

(1) Will retention of records, as proposed, enhance the potential for reducing indoor air quality problems? Will retention of maintenance records, IAQ compliance program records, and records of employee complaints serve as necessary documentation upon which actions and decisions can be made to improve deficiencies found in facility air quality? If so, how will these records serve that purpose?

(2) What length of time should the records required to be established under this proposal be required to be retained? Is OSHA's proposed 3-year retention period reasonable? Why? Why not? Should different retention periods be specified for each particular record, and if so, why?

(3) Is it reasonable to require transfer of records from an employee to a successor employer? What other mechanisms are available to ensure that the facility-specific records remain at the building or facility in the event of tenant turnover?

Dates, Paragraph (i)

Is it feasible for employees to fully implement the provisions of this notice within one year of the effective date, as proposed? Why? Why not? If not, which provisions present difficulties, technologic or economic, with respect to implementation? For which provisions should implementation periods be either decreased or increased and why? To what extent should implementation periods be decreased or increased for particular provisions?

VIII. State Plan Standards

The 25 states and territories with their own OSHA-approved occupational safety and health plans must adopt a comparable standard within six months of the publication date of a final standard. These 25 states are: Alaska, Arizona, California, Connecticut (for public employees only), New York (for state and local government employees only), Hawaii, Indiana, Iowa, Kentucky, Maryland, Michigan, Minnesota, Nevada, New Mexico, North Carolina, Oregon, Puerto Rico, South Carolina, Tennessee, Utah, Vermont, Virginia, Virgin Islands, Washington, and Wyoming. Until such time as a state standard is promulgated, Federal OSHA will provide interim enforcement assistance, as appropriate, in these states.

IX. Federalism

This Notice of Proposed Rulemaking has been reviewed in accordance with Executive Order 12612 (52 FR 41685, October 30, 1987), regarding Federalism. This Order requires that agencies, to the extent possible, refrain from limiting state policy options, consult with states prior to taking any actions which would restrict state policy options, and take such actions only when there is clear constitutional authority and the presence of a problem of national scope. The Order provides for preemption of state law only if there is a clear Congressional intent for the Agency to do so. Any such preemption is to be limited to the extent possible.

Section 18 of the Occupational Safety and Health Act (OSH Act) expresses Congress' intent to preempt state laws that establish occupational safety and health standards on issues on which Federal OSHA has promulgated standards. Under Section 18, a state can avoid preemption, however, if it submits, and obtains Federal approval of a plan for the development of such standards and their enforcement. Therefore states with occupational safety and health plans approved under Section 18 of the OSH Act will be able to develop their own state standards to deal with any special problems which might be encountered in a particular state.

In addition, the Supreme Court has held that Section 18 does not preempt state or local laws of general applicability that do not conflict with OSHA standards and that regulate the conduct of workers and non workers alike. *Gade* v. *National Solid Wastes Management Association*, 112 S. Ct. 2374 (1992). Such laws regulate workers simply as members of the general public. OSHA recognizes that many state and local governments have enacted provisions designed to protect the health of their residents by addressing indoor air quality issues including the presence of ETS. It is OSHA's intent that state and local laws consistent with this standard shall remain in effect to the full extent permissible.

X. Information Collection Requirements

5 CFR part 1320 sets forth procedures for agencies to follow in obtaining OMB clearance for information collection requirements under the Paperwork Reduction Act of 1980, 44 U.S.C. 3501 et seq. This proposed indoor air quality standard requires the employer to allow OSHA access to records. In accordance with the provisions of the Paperwork Reduction Act and the regulations issued pursuant thereto, OSHA certifies that it has submitted the information collection requirements for this proposal to OMB for review under section 3504(h) of that Act.

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TABLE VI–8.—SUMMARY OF COMPLIANCE COSTS FOR PROPOSED OSHA INDOOR AIR QUALITY STANDARD

	Annualized cost ($million)	Recurring cost ($million)	Annual cost ($million)
IAQ written compliance program	$21.1	—	$21.2
IAQ maintenance and operation program	1,281.1	$6,697.4	7,978.5
Information and Training:			
Maintenance workers	0.5	0.8	1.3
All employees	—	—	—
Controls for environmental tobacco smoke [1]	0–68.1	—	0–68.1
Total	1,371.0	6,698.2	8,069.1

[1] Costs incurred are dependent on whether establishments will totally ban smoking or allow smoking in designated areas.
Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, 1994.

The maintenance written plan will also include a description of the equipment to be maintained and the recommended maintenance procedures and frequency of performance. Preferably, the plan should contain the equipment maintenance manuals issued upon completion of facility construction. For establishments in buildings with natural ventilation, employers will develop a plan to assure that windows, doors, vents, stacks and other portals designed or used for natural ventilation are in operable condition.

The cost associated with compiling such information will vary depending on the size of the establishment building, the complexity of the building system, the extent to which such information is already available, and type of occupancy (e.g., single establishment or multi-establishment). In some cases some establishments especially the large ones may already have developed such information. For example, in 1986, IBM initiated a program by first evaluating building design, operation and maintenance and as a result, an IAQ program was devised to include: a model operation/maintenance and IAQ awareness program for building operation/maintenance personnel, an updated building commissioning document and appropriate building lease and contracted operation/maintenance agreements [Ex. 3–904]. There are no data on the number of establishments with IAQ programs. Based on information in the docket, OSHA assumed that 95 percent of all establishments are required to develop the IAQ compliance program information.

In addition, employers are required to: (1) identify a designated person who is given the responsibility of the IAQ compliance program, (2) keep written records of employee complaints of building-related illness and maintenance records, and (3) set up procedures to be utilized during renovation and modeling to minimize degradation of the indoor air quality of employees performing such activities and employees in other areas of the building.

The cost equation for developing the written IAQ compliance program:

$$C_o = E_n \times P_c \times ((W_t \times T_1) + (W_m \times T_2))$$

where

C_o=the cost of developing operation and maintenance information
E_n=the number of establishments
P_c=the percentage of establishments to develop operation and maintenance information (95%)
W_t=the technician wage rate ($15.51 hourly compensation rate)
T_1=the technician time required to compile and develop building system operation and maintenance information (1 hour)
W_m=the managerial wage rate ($30.48 hourly compensation rate)
T_2=the managerial time required to develop some requirements of the written plan (15 minutes)

As presented in Table VI–9, the one time annualized cost of compiling and developing the written IAQ compliance program is $21.2 million.

TABLE VI–9.—COST OF COMPLIANCE FOR DEVELOPING A WRITTEN IAQ PROGRAM

	Total no. of establishments	Annualized first year cost [1] ($million)
Agriculture, forestry, fishing	260,801	$0.91
Mining	22,861	0.08
Construction	642,972	2.24
Manufacturing	389,392	1.35
Transportation	243,769	0.85
Wholesale and retail trade	1,929,891	6.71
Finance, insurance, real estate	526,378	1.83
Services	1,933,750	6.73
Government	135,496	0.47

TABLE VI–9.—COST OF COMPLIANCE FOR DEVELOPING A WRITTEN IAQ PROGRAM—Continued

	Total no. of establishments	Annualized first year cost [1] ($million)
Total	6,085,310	21.17

[1] Based upon 15 minutes of managerial time estimated at $30.48/hr and one hour of technician time estimated at $15.51/hour. Assumes 5 percent existing compliance. Cost is annualized over 10 years at a 10 percent interest rate.
Source: U.S. Department of Labor, OSHA, Office of Regulatory Analysis, 1994.

(b) IAQ Operation and Maintenance Program. The proposed standard requires maintenance and inspection of the building system components that directly affect IAQ. Specifically, the HVAC system should provide at least the outside air ventilation rate based on actual occupancy, building code, mechanical code or ventilation code and that carbon dioxide concentration does not exceed 800 parts per million. In approximately 500 indoor air quality investigations, NIOSH found that the primary cause of indoor air quality problems is inadequate ventilation (52 percent).

Other actions required include: (1) Control of humidity in buildings with mechanical cooling systems, (2) implementing the use of general or local exhaust ventilation where maintenance and housekeeping activities involve use of equipment or products which emit air contaminants in other areas of the facility, (3) maintain mechanical equipment rooms and any non-ducted air plenums or chases in a clean condition.

OSHA recognizes that not every building will have to make all recommended changes to improve operation and maintenance of the HVAC system. In the majority of the cases, some improvements can be accomplished by changing the setting

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(13) Take such remedial measures as the evaluation shows to be necessary.

(e) *Controls for specific contaminant sources.*

(1) *Tobacco smoke.*

(i) In workplaces where the smoking of tobacco products is not prohibited, the employer shall establish designated smoking areas and permit smoking only in such areas;

(ii) The employer shall assure that designated smoking areas are enclosed and exhausted directly to the outside, and are maintained under negative pressure (with respect to surrounding spaces) sufficient to contain tobacco smoke within the designated area;

(iii) The employer shall assure that cleaning and maintenance work in designated smoking areas is conducted only when no smoking is taking place;

(iv) The employer shall assure that employees are not required to enter designated smoking areas in the performance of normal work activities;

(v) The employer shall post signs clearly indicating areas that are designated smoking areas;

(vi) The employer shall post signs that will clearly inform anyone entering the workplace that smoking is restricted to designated areas; and

(vii) The employer shall prohibit smoking within designated smoking areas during any period that the exhaust ventilation system servicing that area is not properly operating.

(2) *Other indoor air contaminants.*

(i) The employer shall implement measures such as the relocation of air intakes and other pathways of building entry, where necessary, to restrict the entry of outdoor air contaminants such as vehicle exhaust fumes, into the building;

(ii) When general ventilation is inadequate to control air contaminants emitted from point sources within workspaces the employer shall implement other control measures such as local source capture exhaust ventilation or substitution.

(3) *Microbial contamination.*

(i) The employer shall control microbial contamination in the building by routinely inspecting for, and promptly repairing, water leaks that can promote growth of biologic agents;

(ii) The employer shall control microbial contamination in the building by promptly drying, replacing, removing, or cleaning damp or wet materials; and

(iii) The employer shall take measures to remove visible microbial contamination in ductwork, humidifiers, other HVAC and building system components, or on building surfaces when found during regular or emergency maintenance activities or during visual inspection.

(4) *Use of cleaning and maintenance chemicals, pesticides, and other hazardous chemicals in the workplace.*

(i) The employer shall assure that these chemicals are used and applied according to manufacturers' recommendations; and

(ii) The employer shall inform employees working in areas to be treated with potentially hazardous chemicals, at least within 24 hours prior to application, of the type of chemicals intended to be applied.

(f) *Air quality during renovation and remodeling.*

(1) *General.* During renovation or remodeling, the employer shall assure that work procedures and appropriate controls are utilized to minimize degradation of the indoor air quality of employees performing such activities and employees in other areas of the building.

(2) *Work plan development.*

(i) Before remodeling, renovation, or similar activities are begun the employer shall meet with the contractor or individual(s) performing the work and shall develop and implement a work plan designed to minimize entry of air contaminants to other areas of the building during and after performance of the work; and

(ii) The work plan shall consider all of the following where appropriate:

(A) Requirements of this standard.

(B) Implementation of means to assure that HVAC systems continue to function effectively during remodeling and renovation activities.

(C) Isolation or containment of work areas and appropriate negative pressure containment.

(D) Air contaminant suppression controls or auxiliary air filtration/cleaning.

(E) Controls to prevent air contaminant entry into the HVAC air distribution system.

(3) *Prior notification of employees who work in the building.*

(i) The employer shall notify employees at least 24 hours in advance, or promptly in emergency situations, of work to be performed on the building that may introduce air contaminants into their work area;

(ii) Notification shall include anticipated adverse impacts on indoor air quality or workplace conditions.

(g) *Employee information and training.*

(1) *The employer shall provide training for maintenance workers and workers involved in building system operation and maintenance which shall include at least the following:*

(i) Training in the use of personal protective equipment (PPE) needed in operating and maintaining building systems;

(ii) Training on how to maintain adequate ventilation of air contaminants generated during building cleaning and maintenance; and

(iii) Training of maintenance personnel on how to minimize adverse effects on indoor air quality during the use and disposal of chemicals and other agents.

(2) *All employees shall be informed of:*

(i) The contents of the standard in this section and its appendices; and

(ii) Signs and symptoms associated with building-related illness and the requirement under paragraphs (d)(12) and (d)(13) of this section directing the employer to evaluate the effectiveness of the HVAC system and to take remedial measures to the HVAC system if necessary, upon receipt of complaints from employees of building-related illness.

(3) *Availability of training material.* The employer shall make training materials developed in response to paragraph (g), including the standard in this section and its appendices, available for inspection and copying by employees, designated employee representatives, the Director, and the Assistant Secretary.

(h) *Recordkeeping.*

(1) *Maintenance records.* The employer shall maintain inspection and maintenance records required to be established under paragraph (d) of this section, which shall include the specific remedial or maintenance actions taken, the name and affiliation of the individual performing the work, and the date of the inspection or maintenance activity.

(2) *Written IAQ compliance program.* The employer shall maintain the written compliance program and plan required to be established under paragraph (c) of this section.

(3) *Employee complaints.* The employer shall maintain a record of employee complaints of signs or symptoms that may be associated with building-related illness required to be established under paragraph (c)(5) of this section. These complaints shall be promptly transmitted to the designated person for resolution.

(4) *Retention of records.* The employer shall retain records required to be maintained under this section for at least the previous three years, except that records required to be maintained under paragraphs (h)(1) and (h)(2) of this section need not be retained for three years if rendered obsolete by the

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(3) The regulation as drafted would require employers generally to prohibit smoking by their customers (such as in bars, restaurants, and stores) where not already banned by a government entity if employees would be exposed to ETS from customer smoking. Comment is requested on the appropriateness of this provision, possible alternatives, and feasibility issues.

Definitions, Paragraph (b)

(1) Is the proposed definition of "air contaminants" sufficiently descriptive to inform employers of the hazards which may adversely affect indoor air quality? If not, what additional information should be included in the definition? Which elements included in the definition are not reflective of hazards which affect indoor air quality?

Can employers reasonably be expected to be able to detect the presence of air contaminants, as defined, and determine whether they present a significant risk of material impairment of employee health? What methods are available to detect indoor air contaminants? What criteria should be used to evaluate the degree of risk that the presence of air contaminants pose to employees?

(2) Is the proposed definition of "building systems" sufficiently descriptive to indicate which systems the employer must attend to in order to assure acceptable indoor air quality? Are the systems listed in the definition those that directly affect indoor air quality? If not, why not? What other systems affect indoor air quality that are not specifically cited in the definition, and how do they influence indoor air quality? How must such systems be maintained and operated in order to assure adequate indoor air quality?

(3) Is the term "building-related illness" sufficiently descriptive and inclusive of the medical conditions that can arise from poor indoor air quality? If not, what other medical conditions should be addressed under the definition and why? Which conditions listed in the definition, if any, should not be considered as "building-related illness" and why?

(4) Is it necessary and appropriate to require employers to authorize a "designated person" to be responsible for ensuring compliance with an indoor air quality standard? Why? Why not? If it is appropriate to require a designated person, what training should designated persons have in order to carry out their responsibilities under the proposed rule? Should the designated person be a person who is a full-time employee who is within the facility each day? Should a designated person be on-site during each shift? Is it unreasonable to expect that due to the complexity of building systems, a single designated person within a facility can successfully oversee and ensure adequate operation of all building systems that affect indoor air quality? Why? Why not? Would it be beneficial for the designated person to receive an inventory of chemical and physical agents used by all employers on site in order to track chemical usage and storage? Information collected could include date of receipt, amount applied or used, where and when in the facility it was used, and how the remainder is stored.

(5) Does the definition of the term "HVAC system" identify all components of HVAC systems which can adversely affect indoor air quality if not properly operated and maintained? What other components should be included and why? What components designated in the definition do not affect indoor air quality and why?

(6) Is the definition of "nonindustrial work environment" sufficiently descriptive to differentiate them from industrial work environments? If not, what other descriptors should be included in the definition? Which types of facilities and establishments proposed under the definition as nonindustrial work environments should not be subject to this standard and why?

(7) Is the definition of "renovation and remodeling" appropriately descriptive of such activities? If not, what modifications to the definition would more reasonably reflect industry view of the characteristics of such activities?

Indoor Air Quality Compliance Program, Paragraph (c)

(1) Is it necessary and appropriate to require employers to establish a written IAQ compliance program in order to assure the adequacy of indoor air quality in nonindustrial work environments? Why? Why not?

(2) If establishment of a written compliance program is necessary, are the informational elements proposed to be developed under this rule appropriate and why? What is their function for successful implementation of the program? Which other written material should be made part of the IAQ compliance program and why?

(3) Which informational elements proposed to be established as part of the IAQ program, if any, are irrelevant to successful building system operation and maintenance? Why?

(4) Which informational elements proposed to be established as part of the IAQ program, if any, are not generally available to the employer and why?

Compliance Program Implementation, Paragraph (d)

(1) Which of the implementation actions proposed under this paragraph are necessary and appropriate for maintenance of acceptable indoor air quality. Why? Which are not? Why not? In this regard, specific comment is particularly sought on the need for the following proposed elements of the implementation program:

(a) Maintenance and operation of the HVAC system to provide at least a required minimum outside air ventilation rate;

(b) Operation of the HVAC during all work shifts;

(c) Use of exhaust ventilation during maintenance and housekeeping activities;

(d) Maintenance of relative humidity to below 60%;

(e) Requiring HVAC system evaluation where CO_2 levels exceed 800 ppm; and

(f) Requiring building system evaluation in response to employee complaints of building related illness.

(g) Should the regulation prohibit the storage of hazardous substances in air transport pathways serving as return air plenums? These areas may include areaways, plenums, chases, corridors, and mechanical rooms serving as return air plenums.

Controls for Specific Contaminant Sources, Paragraph (e)

(1) Will the proposed provisions addressing construction and operation of designated smoking areas assure that employees working outside designated areas will not be exposed to ETS? If so, which of the proposed provisions may be unnecessary to achieve this goal? If not, is it necessary to prohibit smoking within indoor workplaces to eliminate exposure to ETS or can the provisions as proposed be modified, or supplemented to prevent secondary exposure? If it is believed that designated smoking areas will effectively contain tobacco smoke, comment is particularly sought on the appropriateness of requiring designated smoking areas to be enclosed, exhausted directly to the outside and maintained under negative pressure.

(2) Is the proposed provision requiring the use of measures such as local source capture exhaust ventilation or substitution to control air contaminants emitted from point sources where general ventilation is inadequate, feasible or effective?

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establishment and replacement of more recent records, or rendered irrelevant due to HVAC system replacement or redesign.

(5) *Availability.* The records required to be maintained by this paragraph shall be available on request to employees and their designated representative and the Assistant Secretary for examination and copying.

(6) *Transfer of records.* Whenever the employer ceases to do business, records that are required to be maintained by paragraph (h) of this section shall be provided to and retained by the successor employer.

(i) *Dates.* (1) *Effective date.* This section is effective [DATE 60 DAYS FROM PUBLICATION OF THE FINAL RULE]

(2) *Start-up dates.*

Employers shall have implemented all provisions of this standard no later than one year from [THE EFFECTIVE DATE OF THE FINAL RULE].

Appendix A to § _____.____— CARBON DIOXIDE MEASUREMENT PROTOCOL (NONMANDATORY)

Carbon dioxide (CO_2) sampling is one of the measurement tools used to characterize indoor air quality. Indoor CO_2 air concentrations are indicator measures for effectiveness of building ventilation. Elevated carbon dioxide levels can be an indicator of inadequate outside air exchange rates. Carbon dioxide concentrations below 800 ppm generally indicate that the ventilation is adequate for diluting occupant-generated contaminants. The carbon dioxide concentration and the associated outside air ventilation rate offers no confidence as to the adequacy of dilution and removal of other contaminants released in the space. There is also no implication of health effects associated with this level of carbon dioxide, or any implication of a permissible exposure limit. Health effects have been observed in buildings where the carbon dioxide levels were below 800 ppm.

OSHA recommends this procedure:

(1) Design a program of air sampling that includes samples taken: (a) at least every three months to detect the effects of seasonal changes (summer/winter transition seasons); (b) after adjustments have been made to the HVAC system; and (c) at any time there is reason to believe air quality has deteriorated. At least once a year carbon dioxide levels should be monitored when the HVAC system is providing minimum outside air ventilation.

(2) Measure carbon dioxide concentrations late in the morning (about 11:00 am) and late in the afternoon before workers leave for home (about 3:30 pm). These are the times when carbon dioxide levels should be closest to equilibrium levels and should give the best indication of effective air exchange rates. These normal use patterns may be altered by visitor frequency and should be accounted for in the monitoring scheme.

(3) Conduct the sampling at a height of between 3 and 5 feet above the floor, or about the height of employees' heads. Make sure the samples are taken at least 2 feet from where people are breathing. Take the samples at a sufficient distance from any other sources of carbon dioxide so these sources do not affect the measurements.

(4) Select sampling locations in normally-occupied areas where the ventilation mixing would be the least effective. These areas might include corners of a room farthest from supply ducts and exhaust vents, locations surrounded by barriers that might affect air movement, or rooms at the far end of a ventilation supply duct.

(5) Measure the carbon dioxide levels outside the building for comparison with the indoor levels. Average outdoor carbon dioxide levels are typically 300 to 500 ppm.

(6) Use colormetric detector tubes or other direct-reading instruments calibrated and operated according to the manufacturer's instructions for measuring carbon dioxide concentrations.

Take sampling and analytical error into account before comparing results with the 800 ppm benchmark. All measuring devices have a degree of uncertainty associated with the results. An estimate of that uncertainty is called the sampling and analytical error. The uncertainty can be reduced by taking more samples with the same device. Table A–1 can be used to assure 95 percent confidence that the average of the results from a set of detector tube samples is less than 800 ppm. OSHA recommends these following steps:

(1) Calculate the average of the measurements.

(a) Add the detector tube results together.

(b) Divide that total by the number of samples.

(2) Compare the average of the results with the number of samples taken in the second column in the table. If the average is less than the number in the table, there is confidence that the CO_2 levels are less than 800 ppm. Example: Three samples are taken and the results are 650 ppm, 710 ppm, and 680 ppm. The average of these three samples is 680 ppm (2,040 ppm divided by 3). The results indicate confidence that the carbon dioxide levels are less than 800 ppm since the 680 ppm average of the three samples is less than 695 ppm

TABLE A–1.—NUMBER OF SAMPLES TAKEN TO ASSURE 95 PERCENT CONFIDENCE CO_2 CONCENTRATIONS ARE LESS THAN 800 PPM

Number of samples taken	The average [1] (ppm)
2	670
3	695
4	710
5	720
6	725
7	730

[1] The average must be less than.

Table A–2 shows how to determine if the indoor sample results are significantly different from the results taken outdoors. Use this table by following these steps:

(1) Take the same number of samples indoors and outdoors.

(2) Average the results of the outdoor and indoor samples.

(a) Add the outdoor results together and divide by the number of samples taken.

(b) Add the indoor results together and divide by the number of samples taken.

(3) Compare the range of the outdoor and indoor samples.

(a) Subtract the lowest sample result of the outdoor samples from the highest result for the outdoor samples.

(b) Subtract the lowest sample result of the indoor samples from the highest result for the indoor samples.

(4) Calculate *Delta*, which is a term derived by subtracting the difference between the indoor average and the outdoor average and then multiplying that result times 2.

(5) Calculate the *Sum of the Ranges*. Add the outdoor Range and the indoor Range together.

(6) Calculate the *Test Statistic*. Divide *Delta* by the *Sum of the Ranges*.

(7) Compare the *Test Statistic* with the second column in the table below. If the *Test Statistic* is more than the number found in the column, the difference is significant.

Example:

(1) Three samples are taken indoors and three samples are taken outdoors. The results of the outdoor samples are 500 ppm, 580 ppm and 480 ppm. The results of the indoor samples are 650 ppm, 710 ppm, and 680 ppm.

(2) The average of the outdoor samples is 520 ppm (1,560 ppm divided by 3) and the average of the indoor samples is 680 ppm (2,040 ppm divided by 3).

(3) The Range of the outdoor samples is 100 (580 − 480=100) and the Range of the indoor samples is 60 ppm (710 − 650).

(4) "Delta" is 320; (680 − 520)×2=320.

(5) The "Sum of the Ranges" is 160; (100+60)=160.

(6) The "Test Statistic" is 2 (320 divided by 160=2).

(7) Since the "Test Statistic," 2, is greater the 0.974 found in the table for 3 samples, the indoor air levels of carbon dioxide are significantly more than the outdoor air levels.

TABLE A–2.—DETERMINATION OF THE TEST STATISTIC (IF INSIDE CO_2 CONCENTRATION TESTING RESULTS ARE SIGNIFICANTLY DIFFERENT FROM OUTSIDE CONCENTRATIONS (95 PERCENT CONFIDENCE))

Number of samples taken	Test statistic [1]
2	2.322
3	0.974
4	0.644
5	0.493
6	0.405
7	0.347

[1] Test statistic must be more than.

If the indoor sample results show levels that are greater than 800 ppm or that the indoor levels are significantly more than the outdoor levels, initiate actions to investigate

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